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1050 Las Tablas Road, Suite 5 (MRI), Templeton, CA 93465 • (805) 434-1882 • Fax (805) 434-3278

PAGE 2
Patient Name: MCCORNACK DAN E
Date of Exam: 10/12/2004

RIGHT S1 NERVE ROOT.

2. MODERATE RIGHT NEURAL FORAMINAL NARROWING AT THAT LEVEL DUE TO THE DISC PROTRUSION AND FACET JOINT HYPERTROPHY. MILD TO MODERATE LEFT NEURAL FORAMINAL NARROWING AT L5-S1 DUE TO FACET JOINT HYPERTROPHY AND SHORT PEDICLES.
3. FACET JOINT HYPERTROPHY AT L3-4 AND L4-5 AND BULGING DISC AT L4-5 DO NOT APPEAR TO CAUSE NEUROLOGIC COMPROMISE.

James P Cartland MD
JPC /gt

This report has been electronically signed by: James P Cartland MD

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Patient Name: MCCORNACK DAN E
Jacket # 6389

DOB: 02/15/1963
Home # (805)238-5208

Age: 42
Work # (805)239-1550

Physician: GORDON LEMM MD
Telephone #: ( )434-3211

Physician Code: 223
Fax #: 4342019

04/05/2005: CT ABDOMEN AND PELVIS

CLINICAL HISTORY: Adenopathy.

TECHNICAL DATA: Images were obtained on a sixty-four slice Toshiba Aquilion CT scanner. All data was acquired with .5 millimeter collimation. Images of the liver were obtained after administration of oral contrast. Images of the abdomen were obtained during arterial and portal phases after IV contrast administration. Delayed images through the abdomen and pelvis were obtained after a four minute delay. Images are displayed in axial and coronal format.

FINDINGS:

Comparison is made with an earlier study of 4/19/04.

The lung bases are clear. There are no pleural effusions. The liver demonstrates no mass lesion or intrahepatic ductal dilatation. Since the previous examination, there is mildly increased fatty infiltration of the liver. The gallbladder and pancreas are unremarkable. The spleen is mildly enlarged measuring 15 centimeters in maximal AP dimension and 14.4 centimeters in maximal craniocaudal dimension. No splenic mass is identified. The adrenal glands are normal. The kidneys demonstrate no mass or hydronephrosis.

Multiple non pathologic sized retroperitoneal lymph nodes are identified. The largest lymph node in the right periaortic region proximal to the bifurcation measures 12 millimeters and does not appear significantly changed since the previous exam. Multiple smaller left periaortic lymph nodes are also identified. Also seen are multiple non pathologic sized lymph nodes of the mesentery, none measuring over one centimeter.

Small and large bowel caliber is normal. No free fluid. The urinary bladder is unremarkable. There is an umbilical hernia containing fat which is unchanged in appearance. The transverse dimension of this hernia measures 17 millimeters. Scattered sigmoid diverticulosis is present.

IMPRESSION:

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PAGE 2
Patient Name: MCCORNACK DAN E
Date of Exam: 04/05/2005

1. STABLE APPEARANCE OF THE NON PATHOLOGIC SIZED LYMPH NODES IN THE RETROPERITONEUM AND MESENTERY.
2. MILD SPLENOMEGALY, STABLE.
3. UMBILICAL HERNIATION CONTAINING FAT.
4. SIGMOID DIVERTICULOSIS.
5. MILD FATTY INFILTRATION OF THE LIVER WHICH APPEARS MORE PROMINENT SINCE THE PREVIOUS EXAM.

Blake Evernden MD
BE /gt

This report has been electronically signed by: Blake Evernden MD

4/19/05 Patient notified by A Rodman RNA
Mom

5-117

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Patient Name: MCCORNACK DAN E
Jacket # 6389

DOB: 02/15/1963
Home # (805)238-5208

Age: 42
Work # (805)239-1550

Physician: GORDON LEMM MD
Telephone #: ( )434-3211

Physician Code: 223
Fax #: 4342019

08/15/2005: MRI CERVICAL SPINE

CLINICAL HISTORY: Neck pain.

TECHNICAL DATA: The patient was imaged utilizing the Siemens 1.5T Symphony MRI scanner. The protocols executed are as follows: T1 SAG TSE T2 SAG/AX

FINDINGS:

There is straightening of the cervical spine with loss of the normal cervical lordosis. Bone marrow signal is homogeneous without evidence for fracture or suspicious osseous lesion. The posterior fossa structures, as visualized, are unremarkable. No abnormal intrinsic cord signal is detected.

C2-3 and C3-4: Unremarkable.

C4-5: There is minimal posterior bulging of the disc without significant central canal narrowing. Bilateral uncovertebral hypertrophy results in mild bilateral neural foraminal stenosis.

C5-6: Tiny anterior and posterior spurs. Mild desiccation of the disc with a 3 millimeter AP broad-based disc protrusion. This results in moderate central canal compromise. Bilateral uncovertebral hypertrophy results in moderate to severe bilateral neural foraminal stenosis.

C6-7: Small anterior and tiny posterior spurs. Minimal posterior bulging of the disc annulus without significant central canal narrowing. Minimal bilateral uncovertebral hypertrophy results in minimal bilateral neural foraminal stenosis.

C7-T1: No focal disc herniation seen. No significant central canal stenosis. Left-sided uncovertebral hypertrophy results in mild to moderate left neural foraminal stenosis. The right neural foramen is patent.

T1-2 and T2-3: Unremarkable.

IMPRESSION:

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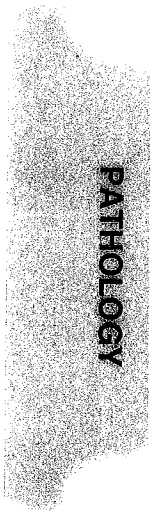
PAGE 2
Patient Name: MCCORNACK DAN E
Date of Exam: 08/15/2005

1. **STRAIGHTENING OF THE CERVICAL SPINE WITH LOSS OF THE NORMAL CERVICAL LORDOSIS.**
2. **TINY POSTERIOR SPUR WITH A BROAD-BASED DISC PROTRUSION AT C5-6 RESULTING IN MODERATE CENTRAL CANAL COMPROMISE WITH MODERATE TO SEVERE BILATERAL NEURAL FORAMINAL STENOSIS.**
3. **TINY POSTERIOR DISC BULGES AT C4-5 AND C6-7 WITHOUT SIGNIFICANT CENTRAL CANAL STENOSIS. MILD NEURAL FORAMINAL NARROWING IS NOTED AT THESE LEVELS.**
4. **MILD TO MODERATE LEFT NEURAL FORAMINAL STENOSIS AT C7-T1.**

Elizabeth M Vogler MD
EMV /gt

This report has been electronically signed by: Elizabeth M Vogler MD


PATHOLOGY

CENTRAL COAST PATHOLOGY CONSULTANTS, INC.
A Medical Group
C.L. Douglas, M.D. J.B. Hannah, M.D. S.B. Jobst, M.D. D.M. Lawrence, M.D.
P.O. BOX 959 Templeton, California 93465
(805) 434-4504 Fax: (805) 434-2913

Patient: McCORNACK, DAN

Accession#: **TWS-96-00964**

Date Coll/Rec'd: 03/11/96 Sex: M DOB: 02/15/63

Physician: **GORDON LEMM, M.D.**

MICROSCOPIC DIAGNOSIS:

Shave biopsy of **SKIN** from "right side of back" showing:

- **AT LEAST JUNCTIONAL NEVUS WITH MILD CYTOLOGIC ATYPIA AND ARCHITECTURAL DISORDER**
- **LESION EXTENDS TO AT LEAST 1 SIDE SURGICAL MARGIN**
- Please see microscopic description.

COMMENT. Previously this entity was known as dysplastic nevus. The treatment for dysplastic nevus is complete but conservative excision, and has not been accomplished here yet.

CLD:clg
03/12/96

Refer to dermatologist
+ send this report

C Douglas MD

Cynthia L. Douglas, M.D. James B. Hannah, M.D. Steven B. Jobst, M.D. David M. Lawrence, M.D.

CENTRAL COAST PATHOLOGY CONSULTANTS, INC.
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P.O. BOX 959 Templeton, California 93465
(805) 434-4504 Fax: (805) 434-2913

Patient: McCORNACK, DAN

Accession#: TWS-96-00964

Date Coll/Rec'd: 03/11/96 Sex: M DOB: 02/15/63

Physician: GORDON LEMM, M.D.

Tissue Received: A) RIGHT SIDE BACK, DARK MOLE

Clinical History: Dark mole, right side of back.

GROSS DESCRIPTION: Received in formalin designated "R side back" is a 3.5 x 2.0 x 1.0 mm oval portion of pale-tan dull membranous tissue with a dark reddish-black macule centrally which in some ways resembles a "blood blister". It does grossly appear completely surrounded by a tan membrane. Longitudinally hemisected into 1 cassette.

CLD:clg
03/11/96

MICROSCOPIC DESCRIPTION: Sections show shave biopsylike portions of skin whose epidermis centrally shows elongation, focal broadening and fusion of its rete. Junctional melanocytes are increased in number, both at the depths and sides of the rete as well as between rete. Diagnostic nesting is not appreciated. Conspicuous is the presence of melanin in melanophages and free in the dermis, both perivascularly and interstitially. Some of the dermal mesenchymal cells appear slightly reactive with vesicular nuclei and small but conspicuous eosinophilic nucleoli. There is no significant upward migration of the junctional cells into the overlying epithelium. The melanocytic process extends to at least 1 side surgical margin. There is fibroplasia in the papillary dermis. In the dermal infiltrate in 1 focus, I am unable to tell if the cells are inflammatory or nevocellular, although they are not nested and some are elongate. Rare junctional nevus cells are enlarged.

DEMGL:0126

CENTRAL COAST PATHOLOGY CONSULTANTS, Inc.

A Medical Group

C. L. Douglas, M.D., Director
B. D. Ragsdale, M.D.
S. B. Jobst, M.D.
R. E. Rocha, M.D.
J. B. Hannah, M.D.
D. M. Lawrence, M.D.
K. F. Lundquist, M.D.
Tel #: (805) 434-4504 Fax #: (805) 434-2913

Test Performed at: Twin Cities Hospital, 1100 Las Tablas Rd., Templeton, California 93465

Patient: McCORNACK, DANIEL SR

Accession #: TWS-03-00869

Sex: Male
DOB: 2/15/63
MRN:

Date Coll/Rec'd: 2/19/03 - 2/20/03

Physician: LEMM, GORDON M.D.

SPECIMEN RECEIVED: **A) Skin specimen**

CLINICAL HISTORY: Lesion, back.

GROSS DESCRIPTION: Received in formalin labeled "back lesion" is a papery-thin shave of white to gray, hair-bearing skin, 3 x 1.5 x 1-mm. One cassette.

CLD/sw:la
2/20/03

MICROSCOPIC DIAGNOSIS: Back skin biopsy:

- DYSPLASTIC COMPOUND MELANOCYTIC NEVUS WITH MODERATE RANDOM CYTOLOGIC ATYPIA OF THE INTRAEPIDERMAL AND DERMAL COMPONENTS, BIOPSY (see Note)

NOTE: The following architectural abnormalities are well-developed: a nested junctional component which radially exceeds a nested dermal component; epithelioid nevomelanocytes bridging adjacent elongate rete; eosinophilic fibroplasia outlining elongate rete; some chronic inflammation with melanophages in superficial dermis. A major criterion of dysplastic nevus, random cytologic atypia, is also present, consisting of occasional melanocytes along the junction with hyperchromatic, irregular nuclear enlargement. In the absence of greater cytologic atypia, pagetoid extension into epidermis or dermal mitotic activity, a benign interpretation is favored.

The general recommendation is for complete excision of dysplastic nevi showing a moderate or higher degree of cytologic atypia. This is because more aggressive histology can sometimes be found adjacent to this picture. Whether or not dysplastic nevi in and of themselves progress to melanoma is a controversial topic, despite the acknowledged fact that 25% of melanomas have contiguous atypical mole histology.

Reference: Ragsdale, B.D., Murphy, G.F. Chapter 17. Tumors of the Skin. In Principles and Practices of Surgical Pathology and Cytology, Third Edition, Editor S.G. Silverberg, Churchill-Livingstone Publishers, pg 415, 1997. BDR:sk

2/26/03 Unsuc - AR for pt to call back

2/27/03 Patient notified by ARodmanRMA Spoke pts wife

Recheck area next month

BRUCE D. RAGSDALE M.D.
Dermatopathologist
Electronically signed 02/24/2003

DEMGL.0124

PLAINTIFFS' EXHIBITS 012473

5-123

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R. E. Rocha, M.D. K. F. Lundquist, M.D. M. V. Frost, M.D. K. L. Ferguson, M.D. A. E. Wilkerson, M.D.

Tel #: (805) 541-6033 Fax #: (805) 541-6116

3701 S. Higuera Street Ste. 200 San Luis Obispo, CA 93401

Patient: MCCORNACK, DANIEL SR

Accession #: TWS-07-10086

Date Coll/Rec'd: 1/10/2007 - 1/10/2007

Sex: M
DOB: 2/15/1963
MRN: T1028685

Physician: **CUSHING, GARY M.D.**
TEMPLETON ENDOSCOPY
LEMM, GORDON M.D.

SPECIMEN RECEIVED: **A) Polyp, GI tract**

CLINICAL HISTORY: 211.3 = Benign neoplasm, large bowel. Procedure: Polypectomy.

GROSS DESCRIPTION: In formalin, labeled "cecal polyp" is one pale pink, curled soft tissue fragment, 3 x 2 x ^ mm. Submitted between sponges, one cassette. Step-cuts are requested. CLD/ph/dl

MICROSCOPIC DIAGNOSIS:

"Cecal polyp" (3 MM GREATEST DIMENSION):
-- COLONIC TYPE MUCOSA WITH SUPERFICIAL CHANGES OF TUBULOVILLOUS ADENOMA

CLD/cbp

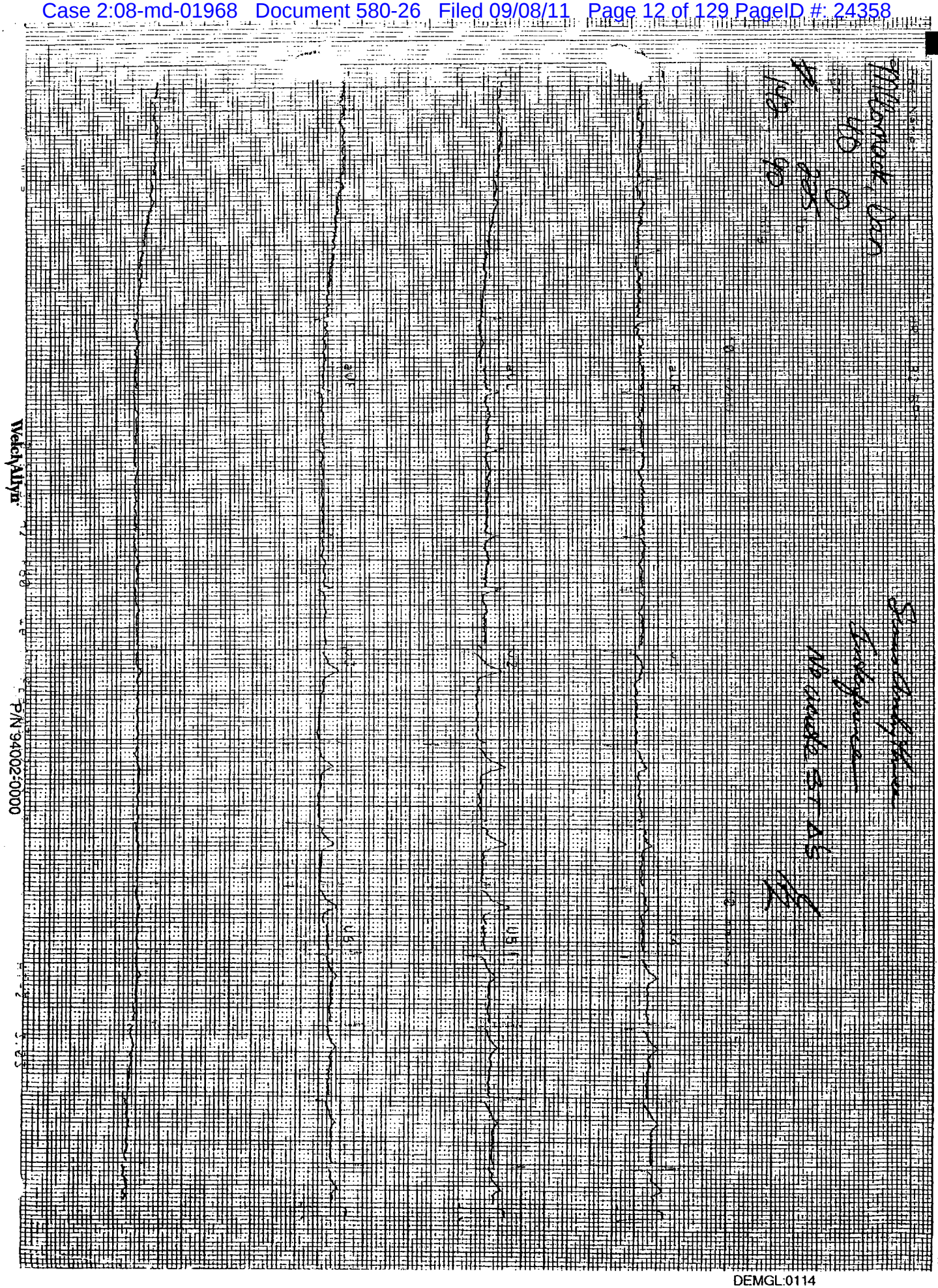
This test was performed at 1100 Las Tablas Road, Templeton, CA 93465

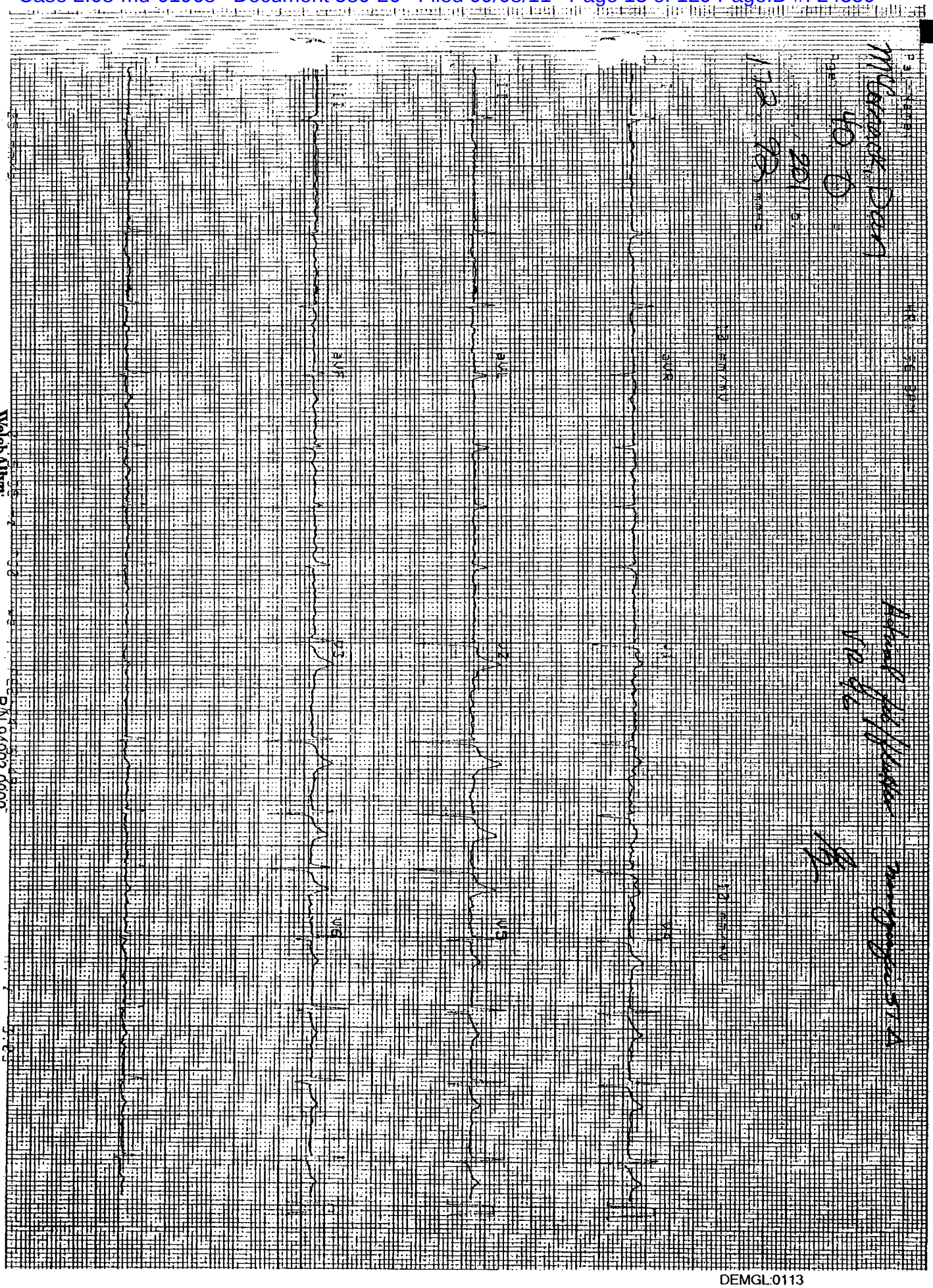
CYNTHIA DOUGLAS M.D.
Pathologist
Electronically signed 1/11/2007 4:42:54PM

5-124

EKG

5-125


Welch Allyn
P/N 94002-0000
DEMGL:0114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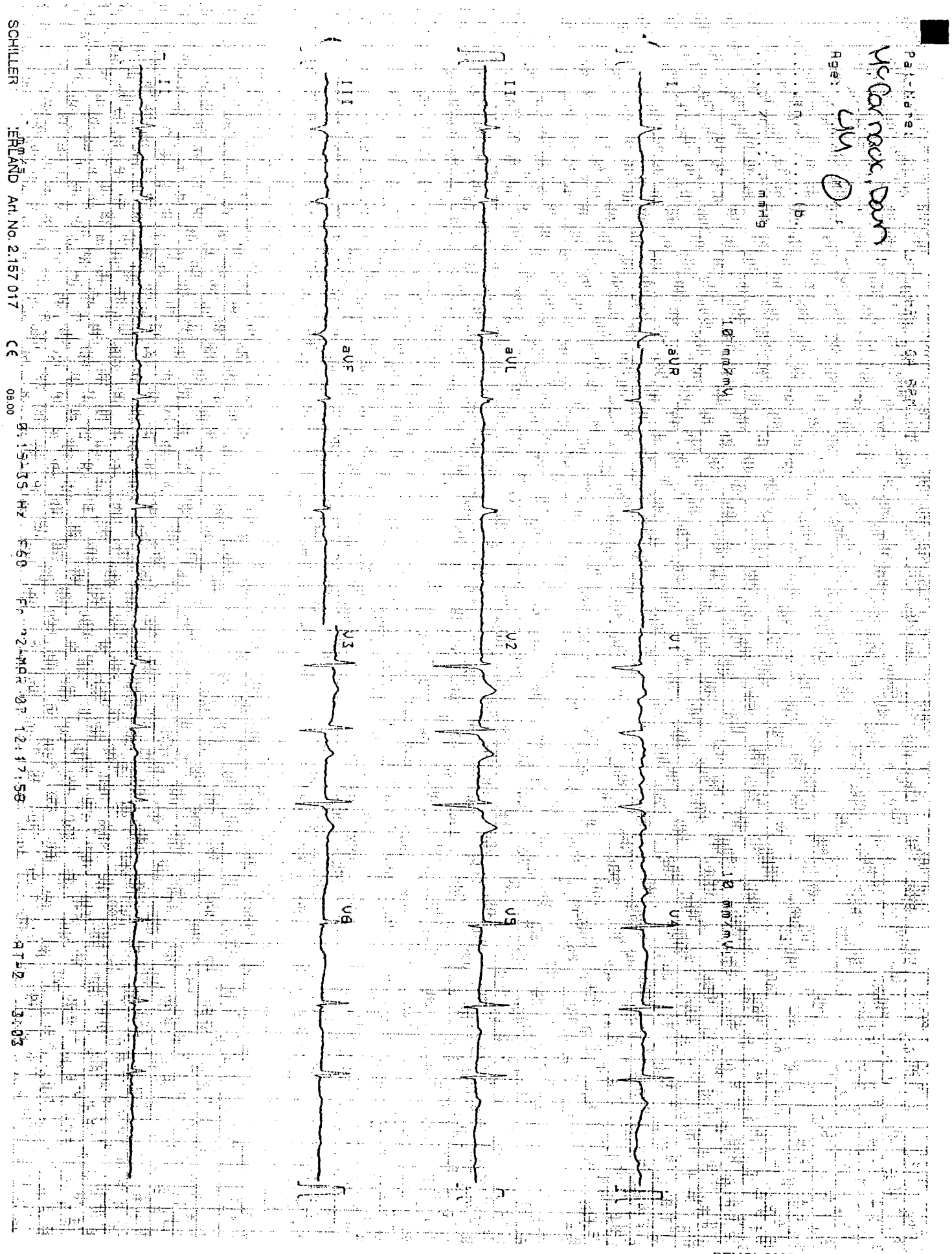
P/N 94002-0000
DEMGL:0113



DEMGL:0093

5-128

RECORDTRAK

651 Allendale Rd.
PO Box 61591
King of Prussia, PA 19406
Phone #: (610) 992-5000
Fax #: (610) 354-8946
www.recordtrak.comRT #:196975 Tag: 2

DANIEL E. MCCORNACK, SR

CASE: DANIEL E. MCCORNACK, SR VS.
ACTAVIS TOTOWA, ET AL
COURT DOCKET: MDL 1968 /
SSN ###-##-7837 D.O.B.: 02/15/1963 D.O.D.: 03/23/2008
PLAINTIFF COUNSEERNST AND MADISON

LOCATION: COASTAL CARDIOLOGY(VON DOLLEN)

Plaintiff Atty:

IN RESPONSE TO RECORDTRAK'S REQUEST FOR THE FOLLOWING:
1. ALL MEDICAL RECORDS IN YOUR POSSESSION. INCLUDE OFFICE AND HAND WRITTEN NOTES, TEST RESULTS, CORRESPONDENCE, QUESTIONNAIRES/HISTORY & RECORDS RECEIVED BY OTHER SHEET. PLEASE BE SURE TO INCLUDE ALL ARCHIVED RECORDS AND ALL RECORDS LOCATED IN STORAGE.
2. SIGNED CERTIFICATION PAGE IS REQUIRED. ***INCLUDING BUT NOT LIMITED TO RECORDS FOR DR. LAWRENCE VON DOLLEN. ***
EDICAL RECORDS AND SIGNED CERTIFICATION(S) ARE ATTACHED.

RECORDTRAK
THE TRACK RECORD OF SUCCESS
• 196975.2 •

DGT.CG01
851 Allendale Road
P. O. Box 61591
King of Prussia, PA 19406
Phone: (800) 220-1291
Fax: (610) 354-8946

August 7, 2009

Re: DANIEL E. MCCORNACK, SR

MEDICAL RECORDS
COASTAL CARDIOLOGY
295 POSADA LANE
SUITE A
TEMPLETON CA 93465

SS #: ###-##-7837
DOB: 02/15/1963 DOD: 03/23/2008
RT File #: 196975 Tag #: 2

Dear Record Custodian:

Attached is an authorization requiring you to furnish *RecordTrak* with the following materials on or before August 17, 2009:

1. ALL MEDICAL RECORDS IN YOUR POSSESSION. INCLUDE OFFICE AND HAND WRITTEN NOTES, TEST RESULTS, CORRESPONDENCE, QUESTIONNAIRES/HISTORY & RECORDS RECEIVED BY OTHER PHYSICIANS. PLEASE ALSO INCLUDE THE PATIENTS INFORMATION SHEET.PLEASE BE SURE TO INCLUDE ALL ARCHIVED RECORDS AND ALL RECORDS LOCATED IN STORAGE.
2. SIGNED CERTIFICATION PAGE IS REQUIRED.
INCLUDING BUT NOT LIMITED TO RECORDS FOR DR. LAWRENCE VON DOLLEN.

Please fax responses along with our request and certifications to RecordTrak at the fax number listed above. If the records are too voluminous to fax, please provide them on CD or mail paper copies to the address listed above.

Before copying and/or invoicing, call or fax *RecordTrak* with a page count and pricing for approval. Please include your federal tax id number on all invoices. Refer to File # 196975 Tag 2 in any correspondence.

Very Truly Yours,

RecordTrak Representative
Phone: (800) 220-1291

IMPORTANT:
*****RESPONSES WILL NOT BE ACCEPTED WITHOUT COMPLETED AND SIGNED CERTIFICATION(S).*****

DEPONENT: COASTAL CARDIOLOGY (TAG 2)
RECORDS PERTAIN TO: DANIEL E. MCCORNACK, SR
RECORDTRAK FILE #: 196975 DATE OF BIRTH: 02/15/1963 SOCIAL SECURITY #: ###-##-7837
RECORD IDENTITY:

DGT.CG01
196975.2

1. ALL MEDICAL RECORDS IN YOUR POSSESSION. INCLUDE OFFICE AND HAND WRITTEN NOTES, TEST RESULTS, CORRESPONDENCE, QUESTIONNAIRES/HISTORY & RECORDS RECEIVED BY OTHER PHYSICIANS. PLEASE ALSO INCLUDE THE PATIENTS INFORMATION SHEET.PLEASE BE SURE TO INCLUDE ALL ARCHIVED RECORDS AND ALL RECORDS LOCATED IN STORAGE. 2. SIGNED CERTIFICATION PAGE IS REQUIRED. ***INCLUDING BUT NOT LIMITED TO RECORDS FOR DR. LAWRENCE VON DOLLEN.***

SECTION I CERTIFICATION OF CUSTODIAN OF RECORDS

I, the undersigned, being the duly authorized custodian of records or other qualified witness, and having the authority to certify the attached records declare the following: the attached records (1) were made at or near the time of the act, event, condition, opinion or diagnosis by a person with knowledge of the matters reflected in the records; (2) were kept in the course of regularly conducted activity; and (3) were created as part of the regular practice of the provider, and that:

A – _____ page(s) of the original records described was made available to the attorney's representative for copying at our place of business.
B – a true, legible and durable copy of 47 pages of the described records was delivered to the attorney's representative.
I DECLARE, UNDER PENALTY OF PERJURY, THAT THE FOREGOING IS TRUE AND CORRECT.

Executed on (date) 9-2-09 at (city,state) San Luis Obispo, Ca
Signature Cassie Penfold Print Name Cassie Penfold
Phone Number 805-546-6190 Department Medical Records

E-mail Address to Forward Requests for Production of Records/Materials: _____

SECTION II CERTIFICATION OF NO RECORDS

A thorough search of our files, carried out under my direction revealed no documents, records or other materials called for in the subpoena or authorization, for the following reason:

- ☐ All records for the time period in question have been destroyed in accordance with our document retention policy which is ___ years.
- ☐ Our records are the same as _____.
- ☐ Original records are in the possession of _____.
- ☐ (other) _____.

I DECLARE, UNDER PENALTY OF PERJURY, THAT THE FOREGOING IS TRUE AND CORRECT.

Executed on (date) _____ at (city,state) _____
Signature _____ Print Name _____
Phone Number _____ Department _____

E-mail Address to Forward Requests for Production of Records/Materials: _____

THIS PAGE MUST BE COMPLETED, SIGNED AND RETURNED.

RECORDS

Patient Chart

Date Printed: 09/02/09

Mccornack, Daniel 555517837 Sex: M Age: 46 DOB: 02/15/1963

Progress Notes

05/11/98
YEARLY F/U

VonDollen
Coastal Cardiology
1105 Las Tablas
Templeton, CA 93465
(805) 434-2262
FAX: (805) 434-2843

Referring Phys.: Gordon Lemm, M.D.

Rx:Current Medications:Current Medications: Rx: DILACOR XR 180MG 1 CAP QD 30 days, 60, Ref: 3
Rx: LANOXIN 0.25MG 1 TAB QD 30 days, 120, Ref: 3
Rx: DILACOR XR 120MG 1 CAP QD 30 days, 30, Ref: 2
Rx: LODINE 1CAP OFF 30 days, 30, Ref: 2

Chronic atrial fibrillation Subjective: No significant chest pain, dyspnea, or syncope. Rare palpitations.

Objective: General Appearance: Alert, oriented X 3, well kempt, conversant - with appropriate affect and mood - no major depression.
Syst. BP 120 Diast. BP 70 P. 64.V2: Wt. 217
Resp: 12 and unlabored
HEENT : Grossly normal external eye and conjunctiva without exudate or hemorrhages. ÿ JVP=4 cm H20. Normal carotid upstroke amplitude and contour bilaterally.
Chest: Grossly normal external thorax. Lungs clear to percussion and auscultation bilaterally. ÿ
Cardiac: PMI normally located without heaves, lifts or thrills. Normal S1and S2 with physiologic splitting with respiration. ÿ Extremities: No peripheral cyanosis, clubbing or edema. ÿ
Assessment: Stable and doing well on current regimen without significant change from last evaluation.ÿ .MP: ATRIAL FIBRILLATIO : 427.31

Plan: Continue current regimen. ÿ Patient Education: Long talk regarding atrial fibrillation - pathophysiology, current treatments - consideration for Holter monitoring to evaluate how fast the ventricular response is at time of greater physical exertion - he notes that he has difficulty keeping up hiking and golfing with his 55 year old father - question if it is just due to deconditioning, atrial fibrillation in gasneral, or due to excesively rapid ventricular response to atrial fibrillation at times of exertion that might explain his poorer physiologic reserve. Over all, he is not interested in any further work up or monitoring at this time, His vision has returned to normal with the discontinuation of the cordarone.
Follow up: 1 year.

SIGNED BY LAWRENCE VON DOLLEN, MD (VON) 05/11/98

Printed using Practice Partner®

Patient Chart

Date Printed: 09/02/09

Mccornack, Daniel 555517837 Sex: M Age: 46 DOB: 02/15/1963

Progress Notes

09/01/99
Atrial Fibrillation 1 yr f/u on Medications

Providers: Vondollen
Coastal Cardiology
295 Posada Suite A
Templeton, CA 93465
(805) 434-2262 Fax (805) 434-2843

Referring Physician: Gordon Lemm, M.D.

Patient Name: Mccornack, Daniel E

Current Medications:
Rx: DILACOR XR 180MG 1 CAP QD 30 days, 90, Ref: 3 ÿ
Rx: LANOXIN 0.25MG 1 TAB bid 30 days, 180, Ref: 3
Rx: DILACOR 120MG 1 TAB qhs 30 days, 90, Ref: 3
Rx: LODINE 1CAP prn 30 days, 30, Ref: 2

Subjective: Mr. McCornack returns for annual follow-up of atrial fibrillation. He has had relatively normal cardiac anatomy and is at low risk cardiac morbidity and mortality. He has a very slightly increased risk of cardioembolic phenomenon from atrial fibrillation, however, with his grossly normal heart and echocardiogram, and his active lifestyle, it would seem that the risk of anticoagulation would probably be greater than his current risk of remaining without anticoagulation. We had a long discussion about current research and current advancement in knowledge about atrial fibrillation and because he remains in a low risk status, we will continue to treat him as he is now with the Dilacor 300 mg p.o. daily and Lanoxin 0.25 mg p.o. b.i.d. He notes that he doesn't have as much stamina as he used to have. He has had no major symptoms other than he just doesn't have as much energy and feels like he has to slow down sooner than he would like to.

Objective:
General Appearance: Alert, oriented X 3, well kept, conversant - with appropriate affect and mood - no major depression.
Syst. BP 130 : Diast. BP 98: P. 80 irr
T : Ht. : Wt. 205 :
Resp: 12 and unlabored
HEENT : Grossly normal external eye and conjunctiva without xanthelasmas, exudate or hemorrhages. Mucous membranes are moist without injection or lesions.
Skin: Warm and dry - no diaphoresis or claminess.
Neck: JVP is normal at 4 cm H20. Normal carotid upstroke amplitude and contour bilaterally.
Chest: Grossly normal external thorax without significant kyphoscoliosis.
Lungs: Clear to percussion and auscultation bilaterally.
Cardiac: PMI normally located without heaves, lifts or thrills. Normal S1and S2
with physiologic splitting with respiration. Soft systolic ejection murmur heard at the left sternal border.
Extremities: No peripheral cyanosis, clubbing or edema.

Assessment: Stable and doing well on current regimen without change from the last evaluation.

Printed using Practice Partner®

Patient Chart

Mccornack, Daniel

Date Printed: 09/02/09

555517837 Sex: M Age: 46 DOB: 02/15/1963

Major Problems: ATRIAL FIB

Plan: Continue current regimen.

Follow up: 1 year.

Lawrence Von Dollen, M.D., F.A.C.C./lt D: 09/02/99 T: 09/03/99

SIGNED BY LAWRENCE VON DOLLEN, MD (VON) 09/03/99

Printed using Practice Partner®

Patient Chart

Date Printed: 09/02/09

Mccornack, Daniel 555517837 Sex: M Age: 46 DOB: 02/15/1963

Progress Notes

02/16/00
Recheck/ AFIB

Providers: VonDollen
Coastal Cardiology
295 Posada Suite A
Templeton, CA 93465
(805) 434-2262 Fax (805) 434-2843

Medical Assistant: DDiioli
Referring Physician: Gordon Lemm, M.D.

Patient Name: Mccomack, Daniel E

Current Medications:
Rx: DILACOR XR 180MG 1 CAP QD 30 days, 90, Ref: 3
Rx: LANOXIN 0.25MG 1 TAB bid 30 days, 180, Ref: 3
Rx: DILACOR 120MG 1 TAB qhs 30 days, 90, Ref: 3
Rx: LODINE 1CAP prn 30 days, 30, Ref: 2

Chief Complaint: He felt terrible on the 1st - hands tingling and going numb, heart doing flip flops, lighthheaded, neck and chest pains - not severe and didn't last long
He doubled up on his lanoxin
he quit chewing copenhagen
Subjective: Mr. McCornack had an episode where he felt his heart was racing and beating much more rapidly, irregularly and forcefully. This occurred around the first of the year without obvious cause. There had been no viral syndrome. He had no change in food or drug habits. He had changed the source of the diltiazem medication but as far as I could tell there were no other major changes. He has gone to drinking more beer with his fishing buddies. He did stop chewing Copenhagen tobacco at that time because of the concern for nicotine stimulation. He has had 2 cups of coffee a day and has reduced that to 1. All in all, he has had no increase in stimulants. There is always a possibility that some of the medication was not working properly. There is always the possibility that he has new anemia or thyroid difficulties.

Objective:
General Appearance: Alert, oriented X 3, well kept, conversant - with appropriate affect and mood - no major depression.
Syst. BP 128 : Diast. BP 78: P. 74
T : Ht. : Wt. 215 :
Resp: 12 and unlabored
HEENT : Grossly normal external eye and conjunctiva without xanthelasmas, exudate or hemorrhages. Mucous membranes are moist without injection or lesions.
Skin: Warm and dry - no diaphoresis or claminess.
Neck: JVP is normal at 4 cm H20. Normal carotid upstroke amplitude and contour bilaterally.
Chest: Grossly normal external thorax without significant kyphoscoliosis.
Lungs: Clear to percussion and auscultation bilaterally.
Cardiac: PMI normally located without heaves, lifts or thrills. Normal S1and S2

with physiologic splitting with respiration.
Extremities: No peripheral cyanosis, clubbing or edema.

Assessment: At the current time he is feeling better overall, including his habits he has changed. An electrocardiogram showed atrial fibrillation with nonspecific ST-T wave changes and moderate ventricular response.

Major Problems: ATRIAL FIB

Plan: We will plan to see him in the near future with Holter monitor if the rate seems to be troublesome again. He states it is calming down over the recent weeks. We will also consider a thyroid panel and other blood tests if he continues to have symptoms. If he does not have symptoms, he will follow-up with Dr. Lemm sometime later this year for his usual physical examination.

Lawrence Von Dollen, M.D., F.A.C.C./lt D: 02/16/00 T: 02/17/00

SIGNED BY LAWRENCE VON DOLLEN, MD (VON) 02/17/00

Patient Chart

Mccornack, Daniel 555517837 Sex: M Age: 46 DOB: 02/15/1963

Date Printed: 09/02/09

Progress Notes

12/18/00
Blood Pressure Check

VonDollen
295 Posada Lane, Suite A
Templeton, CA 93465

Patient Name: MCCORNACK, DANIEL

Chief Complaint: A Fib

S: Patient in for Blood Pressure check.
ATRIAL FIBRILLATIO

Rx: DILACOR 120MG 1 TAB qhs - days, 90, Ref: 3
Rx: DILACOR XR 180MG 1 CAP QD - days, 90, Ref: 3
Rx: LANOXIN 0.25MG 1 TAB bid - days, 180, Ref: 3
Rx:
O:Arm: Left
Syst. BP 160 : Diast BP 90 : P. 88
T : Height : Weight 215

A/Plan: Ok per Dr. VonDollen

BP taken by: MMontgomery

SIGNED BY LAWRENCE VON DOLLEN, MD (VON) 01/06/01

Printed using Practice Partner®

Patient Chart

Date Printed: 09/02/09

Mccornack, Daniel 555517837 Sex: M Age: 46 DOB: 02/15/1963

Progress Notes

04/11/01
: Tiredness and fatigue

Lawrence E. Von Dollen, M.D., F.A.C.C.
Coastal Cardiology
295 Posada Suite A
Templeton, CA 93465
(805) 434-2262 Fax (805) 434-2843

Referring Physician: Gordon Lemm, M.D.

Patient Name: Mccornack, Daniel E

Current Medications:
Rx: DILACOR 120MG 1 TAB qhs - days, 90, Ref: 3
Rx: DILACOR XR 180MG 1 CAP QD - days, 90, Ref: 3
Rx: LANOXIN 0.25MG 1 TAB bid - days, 30, Ref: 0

Chief Complaint: He was promoted and is working harder, but the IRS is taking more with his raise, so he doesn't take home any more than he did before.

Subjective: Mr. McCornack remains NYHA Functional Class I with his lone atrial fibrillation.

Objective:
General Appearance: Alert, oriented X 3, well kept, conversant - with appropriate affect and mood - no major depression.
Syst. BP 114 : Diast. BP 70 P. 80
T : Ht. : Wt. 206
Resp: 12 and unlabored
HEENT : Grossly normal external eye and conjunctiva without xanthelasmas, exudate or hemorrhages. Mucous membranes are moist without injection or lesions.
Skin: Warm and dry - no diaphoresis or claminess.
Neck: JVP is normal at 4 cm H20. Normal carotid upstroke amplitude and contour bilaterally.
Chest: Grossly normal external thorax without significant kyphoscoliosis.
Lungs: Clear to percussion and auscultation bilaterally.
Cardiac: PMI normally located without heaves, lifts or thrills. Normal S1and S2 with physiologic splitting with respiration.
Extremities: No peripheral cyanosis, clubbing or edema.

Assessment: At the current time he is feeling better overall, including his habits he has changed. An electrocardiogram showed atrial fibrillation with nonspecific ST-T wave changes and moderate ventricular response.

Major Problems: ATRIAL FIB

Plan: Do echocardiogram to assess cardiac chamber size and function. Consider review with Dr.

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Patient Chart

Mccornack, Daniel

Date Printed: 09/02/09

555517837 Sex: M Age: 46 DOB: 02/15/1963

Jones after the echocardiogram has been done.

Lawrence Von Dollen, M.D., F.A.C.C./lt D: 04/11/01 T: 04/21/01

SIGNED BY LAWRENCE VON DOLLEN, MD (VON) 04/21/01

Patient Chart

Mccornack, Daniel

Date Printed: 09/02/09
555517837 Sex: M Age: 46 DOB: 02/15/1963

Progress Notes

07/05/01
: Irregular heart beats

Lawrence E. Von Dollen, M.D., F.A.C.C.
Coastal Cardiology
295 Posada Suite A
Templeton, CA 93465
(805) 434-2262 Fax (805) 434-2843

Referring Physician: Gordon Lemm, M.D.

Patient Name: Mccornack, Daniel E

Current Medications:
Rx: DILACOR 120MG 1 TAB qhs - days, 90, Ref: 3
Rx: DILACOR XR 180MG 1 CAP QD - days, 90, Ref: 3
Rx: LANOXIN 0.25MG 1 TAB bid - days, 30, Ref: 0

Chief Complaint: He works a lot with many activities and obligations, but is stable overall without syncope or decreased exercise tolerance .
Subjective: Mr.McCornack remains NYHA Functional Class I with his lone atrial fibrillation. He has had no significant chest pain, palpitations, dyspnea, or syncope.

Objective:
General Appearance: Alert, oriented X 3, well kept, conversant - with appropriate affect and mood - no major depression.
Syst. BP 118 : Diast. BP 78 P. 68
T : Ht. : Wt. 213
Resp: 12 and unlabored
HEENT : Grossly normal external eye and conjunctiva without xanthelasmas, exudate or hemorrhages. Mucous membranes are moist without injection or lesions.
Skin: Warm and dry - no diaphoresis or claminess.
Neck: JVP is normal at 4 cm H20. Normal carotid upstroke amplitude and contour bilaterally.
Chest: Grossly normal external thorax without significant kyphoscoliosis.
Lungs: Clear to percussion and auscultation bilaterally.
Cardiac: PMI normally located without heaves, lifts or thrills. Normal S1and S2 with physiologic splitting with respiration.
Extremities: No peripheral cyanosis, clubbing or edema.

Assessment: At the current time he is feeling pretty good overall. His Holter monitor showed atrial fibrillation with slightly fast ventricular response on Dilacor 180 po QAM and 120mg po QPM. His echocardiogram showed normal cardiac anatomy and function - the LA was 41mm.

Major Problems: ATRIAL FIB

Plan: Continue the current meds, but increase the dilacor to 300mg po QAM and 180mg po QPM. Check back in 1 month. Consider increasing the digoxin if the dig level allows and the heart rate

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remains elevated. Review with Dr. Jones - no major innovations that are likely to change management.

SIGNED BY LAWRENCE VON DOLLEN, MD (VON) 07/05/01

Patient Chart

Mccornack, Daniel

Date Printed: 09/02/09
555517837 Sex: M Age: 46 DOB: 02/15/1963

Progress Notes

08/07/01
: Irregular heart beats

Lawrence E. Von Dollen, M.D., F.A.C.C.
Coastal Cardiology
295 Posada Suite A
Templeton, CA 93465
(805) 434-2262 Fax (805) 434-2843

Referring Physician: Gordon Lemm, M.D.

Patient Name: Mccornack, Daniel E

Current Medications:
Rx: DILACOR 300MG 1 qd - days, , Ref: 6
Rx: DILACOR XR 180MG 1 qhs - days, , Ref: 6
Rx: LANOXIN 0.25MG 1 bid - days, 90, Ref: 3

Chief Complaint: Variable pains in his feet - no evidence of TIA's or other cardioembolic phenomena.
Subjective: He is seeing Dr Yamagata for unexplained pains in his right and left feet. There has been no paresis, back pain, or sensory deficits. He has a lot of stress aas he works a lot with many activities and obligations, but is stable overall without syncope or decreased exercise tolerance .
Mr.McCornack remains NYHA Functional Class I with his lone atrial fibrillation. He has had no significant chest pain, palpitations, dyspnea, or syncope.

Objective:
General Appearance: Alert, oriented X 3, well kept, conversant - with appropriate affect and mood - no major depression.
Syst. BP 120 : Diast. BP 88 P. 70
T : Ht. : Wt. 209
Resp: 12 and unlabored
HEENT : Grossly normal external eye and conjunctiva without xanthelasmas, exudate or hemorrhages. Mucous membranes are moist without injection or lesions.
Skin: Warm and dry - no diaphoresis or claminess.
Neck: JVP is normal at 4 cm H20. Normal carotid upstroke amplitude and contour bilaterally.
Chest: Grossly normal external thorax without significant kyphoscoliosis.
Lungs: Clear to percussion and auscultation bilaterally.
Cardiac: PMI normally located without heaves, lifts or thrills. Normal S1and S2 with physiologic splitting with respiration.
Extremities: No peripheral cyanosis, clubbing or edema.

Assessment: At the current time he is feeling pretty good overall. His Holter monitor showed atrial fibrillation with slightly fast ventricular response on Dilacor 180 po QAM and 120mg po QPM, so his dose was increased to 300mg po QAM and 180mg po QPM with reduction in the heart rate in the 70's. His echocardiogram showed normal cardiac anatomy and function - the LA was 41mm.

Major Problems: ATRIAL FIB

Patient Chart

Date Printed: 09/02/09

Mccornack, Daniel 555517837 Sex: M Age: 46 DOB: 02/15/1963

Hyperuricemia

Plan: Continue the dilacor 300mg po QAM and 180mg po QPM. Check back in 6 months. Continue the current dose of digoxin since his level is normal at 1.7 on the current dose with good ventricular response.

SIGNED BY LAWRENCE VON DOLLEN, MD (VON) 08/07/01

REVISED BY LAWRENCE VON DOLLEN, MD (VON) 08/07/01

Printed using Practice Partner®

Patient Chart

Date Printed: 09/02/09

Mccornack, Daniel 555517837 Sex: M Age: 46 DOB: 02/15/1963

Progress Notes

02/25/02
: Irregular heart beats

Lawrence E. Von Dollen, M.D., F.A.C.C.
Coastal Cardiology
295 Posada Suite A
Templeton, CA 93465
(805) 434-2262 Fax (805) 434-2843

Referring Physician: Gordon Lemm, M.D.

Medical Assistant: KRosson

Patient Name: McCornack, Daniel E

Current Medications:
Rx: DILACOR 300MG 1 qd - days, , Ref: 6
Rx: DILACOR XR 180MG 1 qhs - days, , Ref: 6
Rx: LANOXIN 0.25MG 1 bid - days, 90, Ref: 3
Rx: ALLOPURINOL 100MG 2 qd - days, , Ref: 0

Chief Complaint: Palpitations.
Subjective: He really cannot tell that he has atrial fibrillation any more. He has a lot of stress as he works a lot with many activities and obligations, but is stable overall without syncope or decreased exercise tolerance .
Mr.McCornack remains NYHA Functional Class I with his lone atrial fibrillation. He has had no significant chest pain, palpitations, dyspnea, or syncope. He only rarely has had the variable pains in his feet that he had at the time of the last visit; he has had no evidence of TIA's or other cardioembolic phenomena.

Objective:
General Appearance: Alert, oriented X 3, well kept, conversant - with appropriate affect and mood - no major depression.

Bp: 142/74, Pulse: 88
Weight: 218

HEENT : Grossly normal external eye and conjunctiva without xanthelasmas, exudate or hemorrhages. Mucous membranes are moist without injection or lesions.
Skin: Warm and dry - no diaphoresis or claminess.
Neck: JVP is normal at 4 cm H20. Normal carotid upstroke amplitude and contour bilaterally.
Chest: Grossly normal external thorax without significant kyphoscoliosis.
Lungs: Clear to percussion and auscultation bilaterally.
Cardiac: PMI normally located without heaves, lifts or thrills. Normal S1and S2 with physiologic splitting with respiration.
Extremities: No peripheral cyanosis, clubbing or edema.

Patient Chart

Date Printed: 09/02/09

Mccornack, Daniel 555517837 Sex: M Age: 46 DOB: 02/15/1963

Assessment: At the current time he is feeling pretty good overall. He has had no significant chest pain, palpitations, dyspnea, or syncope.
Major Problems: ATRIAL FIB
Hyperuricemia

Plan: Continue the current regimen

SIGNED BY LAWRENCE VON DOLLEN, MD (VON) 02/25/02

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Patient Chart

Date Printed: 09/02/09

Mccornack, Daniel 555517837 Sex: M Age: 46 DOB: 02/15/1963

Progress Notes

04/21/03
Irregular heart beats

Lawrence E. Von Dollen, M.D., F.A.C.C.
Coastal Cardiology
295 Posada Suite A
Templeton, CA 93465
(805) 434-2262 Fax (805) 434-2843

Referring Physician: Gordon Lemm, M.D.

Medical Assistant: BMinter,RN

Patient Name: McCornack, Daniel E

Current Medications:
Rx: DILACOR 300MG 1 qd - days, , Ref: 6
Rx: DILACOR XR 180MG 1 qhs - days, , Ref: 6
Rx: LANOXIN 0.25MG 1 bid - days, 90, Ref: 3
Rx: ALLOPURINOL 100MG 2 qd - days, , Ref: 0

Chief Complaint: Palpitations.
Subjective: He has a lot of stress as he works a lot with many activities and obligations, but is stable overall without syncope or deercased exercise tolerance .
He really cannot tell that he has atrial fibrillation, and he remains NYHA Functional Class I with his lone atrial fibrillation. Mr.McCornack has had no significant chest pain, palpitations, dyspnea, or syncope. He has had no evidence of TIA's or other cardioembolic phenomena.

Objective:
General Appearance: Alert, oriented X 3, well kept, conversant - with appropriate affect and mood - no major depression.
Bp: 148/94, Pulse: 88
Weight: 220
HEENT: Grossly normal external eye and conjunctiva without xanthelasmas, exudate or hemorrhages. Mucous membranes are moist without injection or lesions.
Skin: Warm and dry - no diaphoresis or claminess.
Neck: JVP is normal at 4 cm H20. Normal carotid upstroke amplitude and contour bilaterally.
Chest: Grossly normal external thorax without significant kyphoscoliosis.
Lungs: Clear to percussion and auscultation bilaterally.
Cardiac: PMI normally located without heaves, lifts or thrills. Normal S1and S2
with physiologic splitting with respiration.
Extremities: No peripheral cyanosis, clubbing or edema.

Assessment: At the current time he is feeling good overall, but he has turned forty and is not as quick or energetic as he was in years gone by - he notices it in community athletics . He has had no significant chest pain, palpitations, dyspnea, or syncope.
Major Problems: ATRIAL FIB

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Patient Chart

Mccornack, Daniel

Date Printed: 09/02/09
555517837 Sex: M Age: 46 DOB: 02/15/1963

Hyperuricemia

Plan: Continue the current regimen.

SIGNED BY LAWRENCE VON DOLLEN, MD (VON) 04/22/03

Patient Chart

Date Printed: 09/02/09

Mccornack, Daniel 555517837 Sex: M Age: 46 DOB: 02/15/1963

Progress Notes

04/21/04
Irregular heart beats

Lawrence E. Von Dollen, M.D., F.A.C.C.
Coastal Cardiology
295 Posada Suite A
Templeton, CA 93465
(805) 434-2262 Fax (805) 434-2843

Referring Physician: Gordon Lemm, M.D.

Medical Assistant: jrussell
Patient Name: McCornack, Daniel E

Current Medications:
Rx: LANOXIN 0.25MG 1 bid - days, 90, Ref: 3
Rx: ALLOPURINOL 300MG 1 qd - days, , Ref: 0
Rx: PROTONIX 40mg 1 qd - days, , Ref: 0

Chief Complaint: Palpitations

Subjective: Mr.McCornack has very much reconciled and really has no side effects to atrial fibrillation. He has had many stresses recently with regard to job difficulties, also abdominal bloating discomfort with is diagnosed as having perhaps prostate infection. There is also a question of lymphadenopathy, which raises a potential diagnosis of greater concern in terms of malignancy or some other cause of lymphadenopathy. He is being treated for prostatitis and see if this will clear things up. In the meantime, he has had aches and pains over his chest, over his neck, his arms that will last sometimes days at a time. We reviewed the common symptom complex of coronary artery disease versus stress reaction versus musculoskeletal. All things considered, he really has none of the major criteria of symptoms that would go along with ischemic heart disease, nor is there a cardioembolic phenomenon from his atrial fibrillation. He does occasionally has sweatiness and coldness of his hands. This is not particularly associated with any events or times or activities. He has lost weight on the Adkins diet. He feels better, more active. He does feel his age, he is over 40 now.

Objective:
General Appearance: Alert, oriented X 3, well kept, conversant - with appropriate affect and mood - no major depression.
BP: 132/88, Pulse: 84
Height: 70", Weight: 197
HEENT: Grossly normal external eye and conjunctiva without xanthelasmas, exudate or hemorrhages. Mucous membranes are moist without injection or lesions.
Skin: Warm and dry - no diaphoresis or claminess.
Neck: JVP is normal at 4 cm H20. Normal carotid upstroke amplitude and contour bilaterally.
Chest: Grossly normal external thorax without significant kyphoscoliosis.
Lungs: Clear to percussion and auscultation bilaterally.
Cardiac: PMI normally located without heaves, lifts or thrills. Normal S1 and S2 with physiologic splitting with respiration.
Extremities: No peripheral cyanosis, clubbing or edema.

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Patient Chart

Date Printed: 09/02/09

Mccornack, Daniel 555517837 Sex: M Age: 46 DOB: 02/15/1963

Assessment: Stable cardiovascular status by all indications.

Major Problems: ATRIAL FIB
Hyperuricemia

Plan: Follow-up in 6 months or a year as requested.

Rx: PROTONIX 40mg 1 qd , , Ref: 0

Lawrence Von Dollen, M.D., F.A.C.C./lt D&T: 04/22/04

SIGNED BY LAWRENCE VON DOLLEN, MD (VON) 04/22/04

Printed using Practice Partner®

Patient Chart

Mccornack, Daniel

Date Printed: 09/02/09
555517837 Sex: M Age: 46 DOB: 02/15/1963

Progress Notes

11/27/06 : 11:44am
Office Visit
PV: Von
Coastal Cardiology
295 Posada Suite A
Templeton, CA 93465
(805) 434-2262 Fax (805) 434-2843

Refering Physician: Gordon Lemm, M.D.

Medical Assistant: Yleppelman,CMA

Patient Name: McCornack, Daniel E

Rx: DILACOR 300MG 1 qd
Rx: DILACOR XR 180MG 1 qhs
Rx: LANOXIN 0.25MG 1 bid
Rx: ALLOPURINOL 300MG 1 qd
Rx: ASPIRIN-COATED 325MG 1 qd
Rx: PREVACID 30MG 1 qd

Chief Complaint: Palpitations

Subjective: Mr. McCornack has occasional skipped heartbeats and palpitations. He has had no dizziness or lightheadedness. For the most part the rate of atrial fibrillation seems to be relatively stable with the current Dilacor and Lanoxin. In the past year or so, he has been worked up for lymphoma by Dr. Lemm and also was determined to have one of the HLA B-27 associated syndrome. More recently he has had night sweats and diaphoresis. He has also had the sensation of cold extremities under different circumstances. We do not have a good explanation for that. He has also raised the topic with Dr. Lemm. At this point there are no obvious cardiac causes other than tachy or bradyarrhythmias. He feels that he has a good idea about whether his heart is going fast or slow. We did talk about doing monitoring to see if that might be an explanation for his episodes.

He notes that when he goes out doing active physical things with his friends, he is no longer able to keep up with them as easily as he has in the past. He questions whether he might be considered for an electrophysiology treatment of atrial fibrillation. We will check his cardiac ultrasound, laboratory and then address the issue with Dr. Winkle now that more progress has been made in treatment of atrial fibrillation with electrophysiologic techniques.

Objective:
General Appearance: Alert, oriented X 3, well kept, conversant - with appropriate affect and mood - no major depression.
BP: 140/88, Pulse: 81
Height: 5'10, Weight: 229
HEENT : Grossly normal external eye and conjunctiva without xanthelasmas, exudate or hemorrhages. Mucous membranes are moist without injection or lesions.
Skin: Warm and dry - no diaphoresis or clamminess.
Neck: JVP is normal at 4 cm H20. Normal carotid upstroke amplitude and contour bilaterally.
Chest: Grossly normal external thorax without significant kyphoscoliosis.
Lungs: Clear to percussion and auscultation bilaterally.
Cardiac: PMI normally located without heaves, lifts or thrills. Normal S1and S2 with physiologic splitting with respiration.

Printed using Practice Partner®

Patient Chart

Date Printed: 09/02/09

Mccornack, Daniel 555517837 Sex: M Age: 46 DOB: 02/15/1963

Extremities: No peripheral cyanosis, clubbing or edema.

Assessment: Stable cardiovascular status by all indications.
Major Problems: ATRIAL FIB
Hyperuricemia
HLA B-27

Plan: We will see him back in a year or as requested.

Rx: PREVACID 30MG 1 qd , , Ref: 0
Rx: ASPIRIN-COATED 325MG 1 qd , , Ref: 0

Lawrence Von Dollen, M.D., F.A.C.C./lt D&T: 11/27/06

SIGNED BY LAWRENCE VON DOLLEN (VON) 11/27/2006 03:45PM

REVISED BY LAWRENCE VON DOLLEN (VON) 11/28/2006 09:20AM

Printed using Practice Partner®

Patient Chart

Date Printed: 09/02/09

Mccornack, Daniel 555517837 Sex: M Age: 46 DOB: 02/15/1963

Progress Notes

07/13/07 : 12:05pm
Office Visit
VON

Lawrence Von Dollen, M.D., F.A.C.C.
Coastal Cardiology
295 Posada Suite A
Templeton, CA 93465
(805) 434-2262 Fax (805) 434-2843

Refering Physician: Gordon Lemm, M.D.

Medical Assistant: P.Callison,RN

Patient Name: McComack, Daniel E

Rx: DILACOR 300MG 1 qd
Rx: DILACOR XR 180MG 1 qhs
Rx: LANOXIN 0.25MG 1 bid
Rx: ALLOPURINOL 300MG 1 qd
Rx: ASPIRIN-COATED 325MG 1 qd
Rx: PREVACID 30MG 1 qd

Chief Complaint: Palpitations

Subjective: Mr. McComack returns with motivation to try to restore sinus rhythm. He has address the issue wit Dr. Winkle now that more progress has been made in treatment of atrial fibrillation with electrophysiologic techniques.
He had night sweats and diaphoresis and has been worked up for lymphoma by Dr. Lemm and also was determined to have one of the HLA B-27 associated syndrome. He has also had the sensation of cold extremities under different circumstances. We do not have a good explanation for that. He notes that when he goes out doing active physical things with his friends, he is no longer able to keep up with them as easily as he has in the past.

Objective:
General Appearance: Alert, oriented X 3, well kept, conversant - with appropriate affect and mood - no major depression.
BP: 136/80, Pulse: 77
Height: 70", Weight: 224
HEENT : Grossly normal external eye and conjunctiva without xanthelasmas, exudate or hemorrhages. Mucous membranes are moist without injection or lesions.
Skin: Warm and dry - no diaphoresis or clamminess.
Neck: JVP is normal at 4 cm H20. Normal carotid upstroke amplitude and contour bilaterally.
Chest: Grossly normal external thorax without significant kyphoscoliosis.
Lungs: Clear to percussion and auscultation bilaterally.
Cardiac: PMI normally located without heaves, lifts or thrills. Normal S1and S2 with physiologic splitting with respiration.
Extremities: No peripheral cyanosis, clubbing or edema.

Assessment: Generally stable cardiovascular status but NYHA Functional Class I-II with DOE and chronic atrial fibrillation with moderate ventricular response.

Patient Chart

Date Printed: 09/02/09

Mccornack, Daniel 555517837 Sex: M Age: 46 DOB: 02/15/1963

Major Problems: ATRIAL FIB
Hyperuricemia
HLA B-27

Plan: Before proceeding with electrophysiological evaluation with possible radiofrequency ablation in attempts to treat atrial fibrillation, we will try to convert from atrial fibrillation to sinus rhythm to see if symptomatically he is changed by restoration of sinus rhythm. Toward that end, we will initiate Coumadin anticoagulation and then proceed with cardioversion once he has had therapeutic INR for at least 3 weeks. He has been intolerant of amiodarone in the past, consider Rythmol, consider dofetilide if appropriate. He remains on Lanoxin and Dilacor. We will be in touch with Dr. Winkle with regard to his workup and care.

Lawrence Von Dollen, M.D./F.A.C.C./lt D: 08/06/07 T: 08/16/07

SIGNED BY LAWRENCE VONDOLLEN (VON) 08/16/2007 09:53AM

Printed using Practice Partner®

Patient Chart

Date Printed: 09/02/09

Mccornack, Daniel 555517837 Sex: M Age: 46 DOB: 02/15/1963

Progress Notes

11/29/07 : 08:48am
AFIB
Medical Assistant: Sakisha Alexander, CMA
Patient Name: MCCORNACK, DANIEL
02/15/63
401:VON

Coastal Cardiology
1941 Johnson Avenue Suite 101
San Luis Obispo, CA 93401
(805) 782-8844
FAX: (805) 782-8859

Referring Phys.: Gordon Lemm, M.D.
Last visit with PMD:3 months ago
Coastal Cardiology Provider/Last visit/Reason:
Cardiologist: Lawrence Von Dollen, M.D., F.A.C.C.
1941 Johnson Avenue Suite 101
San Luis Obispo, CA 93401
(805) 782-8844 FAX (805) 782-8859

Chief Complaint: AFIB

Subjective: 44 year old patient in the office today for follow up AFIB. He states he is back from his hunting trips and would like to discuss medical therapy plan. He has seen Dr Winkle in June 07 to discuss ablation, but has opted not to proceed. He wanted to hold off on an ablation due to the hunting season. He states he is unclear what to proceed with vs doing nothing. He states he feels relatively well, with some exception to doing exertional exercise. He is able to hike while hunting, but does have DOE - he states he isn't clear whether it is related to decreased stamina vs AFIB. He denies of any chest pain/pressure, edema, CHF symptoms, or dizziness.

Allergies: SULFA, AMPICILLIAN

Current Medications:
Rx: DILACOR 300MG 1 qd
Rx: DILACOR XR 180MG 1 qhs
Rx: LANOXIN 0.25MG 1 bid
Rx: ALLOPURINOL 300MG 1 qd
Rx: ASPIRIN-COATED 325MG 1 qd
Rx: PREVACID 30MG 1 qd

Review of Systems: unchanged compared with last visit
PFSH: Reviewed and unchanged compared with last visit
DIAGNOSTIC STUDIES COMPLETED:
ECHO: completed on 12/14/2006
Dr Winkles note 06/2007: See chart

ECG: completed on 07/13/2007
HOLTER: completed on 06/12/2001
Major Problem List:
ATRIAL FIBRILLATIO
HLA B-27

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Patient Chart

Date Printed: 09/02/09

Mccornack, Daniel 555517837 Sex: M Age: 46 DOB: 02/15/1963

Objective:
Syst. BP 110 : Diast BP 72 : P. 79
T : Height 5'10" : Weight 224
Recheck BP:112/68 Pain: 0/10, RR: 12 and unlabored
General Appearance: The patient is a male who appears stated age in no acute distress.
HEENT: The patient is normcephalic. Grossly normal external eye and conjunctiva without xanthelasmas, exudate or hemorrhages. Grossly normal oropharynx with dentition in reasonable repair.
NECK: The neck is supple and trachea midline. Thyroid nonpalpable without masses noted. No lymphadenopathy. Jugular venous pressure is less than 5 cm. Normal carotid upstroke, amplitude and contour bilaterally. No bruits or transmitted murmurs are noted.
SKIN: Pink, warm and dry - no diaphoresis or clamminess. No rashes noted.
CHEST: The chest is symmetrical with no chest wall abnormality. Normal respiratory effort noted.
CARDIAC: Irregular rate and rhythm with normal S1 and physiologically split S2. The PMI is not palpable not displaced. No palpable lifts, heaves or thrills are present. No gallops, murmurs, clicks or rubs are noted.
LUNGS: No use of accessory muscles or retractions. Lungs are clear to auscultation and percussion.
ABDOMEN: Abdomen is nontender, nondistended, soft without scars noted. No hepatosplenomegaly or masses palpable. Bowel sounds normal. There are no aortic or renal bruits noted. The aortic pulsations are normal not felt.
EXTREMITIES: Extremities are warm without cyanosis, clubbing or edema. +2 peripheral pulses intact.
NEURO: Patient alert and oriented. Grossly nonfocal, appropriate mood and affect. Normal gait present.

Assessment:
Major Problem: Atrial Fibrillation - rate controlled
Major Problem: ASA therapy

Plan:
1. Discussed with patient the options for treatment for AFIB. Anticoagulation and cardioversion with antiarrhythmics vs ablation by Dr Winkle, vs do nothing. He is very unclear what he would like to do at this time. He will continue ASA on a daily basis.
2. I will discuss with Dr VonDollen regarding his thoughts about his options.
3. I will contact patient after speaking with Dr VonDollen

Patient Education: Indications, benefits and complications of anticoagulation were discussed. Well balanced diet, low salt, low fat. Stressed the importance of regular exercise. Please contact the office immediately if you need refills or difficulty receiving, paying for, or understanding the use of medications. dietary

Follow up: I will call patient

I spent 45 minutes with the patient; greater than 50% of the office visit was spent counseling and coordination of care.

SIGNED BY JESSICA MALONE (401) 11/30/2007 02:54PM

CO-SIGNED BY LAWRENCE VONDOLLEN (VON) 03/04/2008 09:57PM

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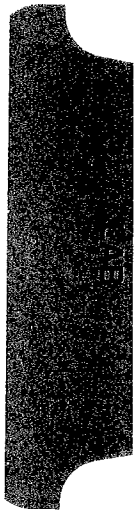

5-157

Patient Chart

Date Printed: 09/02/09

Mccornack, Daniel 555517837 Sex: M Age: 46 DOB: 02/15/1963

Laboratory Data

11/14/02
LAB RESULTS
Time collected: 0840
Patient Name: McCornack, Daniel
Lab Performed: Twin Cities CommunityHospital
Data entered by: kbrown
Date received: 111502
Date entered: 111502
VONDOLLEN

Outside Ordering Physician: Lemm, G

HFP: Completed
TBIL: 1.0
GOT: 33
GPT: 82****
ALK: 76
ALB: 4.6
CMP: Completed
GLU: 130*****
NA: 141
K: 4.2
CL: 100
CO2: 31.1***
AGAP: 14.1
BUN: 24*****
CR: 1.4
UR: 8.2*****
CA: 10.5****
TP: 7.6
DRG SERUM : Completed
DIG: 1.5

SIGNED BY LAWRENCE VON DOLLEN, MD (VON) 12/16/02

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Patient Chart

Mccornack, Daniel

Date Printed: 09/02/09
555517837 Sex: M Age: 46 DOB: 02/15/1963

Laboratory Data

02/20/04
LAB RESULTS
Time collected: 0820
Patient Name: Mccornack, Daniel
DOB: 02/15/63
Lab Performed: Twin Cities Community Hospital
Data entered by: dpunches
Date received: 022304
Date entered: 022304
VONDOLLEN

Outside Physician: Lemm

LIP2: Completed
TRIG: 229***
CHOL: 254***
HDL: 43.5
LDL: 165***
TC/HD: 5.8***
HFP: Completed
TBIL: 0.6
GOT: 21
GPT: 47
ALK: 61
ALB: 4.7
CMP: Completed
GLU: 109
NA: 143
K: 4.4
CL: 102
CO2: 32.6***
AGAP: 12.8
BUN: 27***
CR: 1.1
CA: 9.8
TP: 6.9
ENDOCRINE : Completed
TSH: 3.24
T4: 7.5
CBC : Completed
WBC: 9.5
RBC: 5.45
HGB: 16.9
HCT: 48.9
MCV: 89.8
MCHC: 34.6
PLT: 167

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Patient Chart

Date Printed: 09/02/09

Mccornack, Daniel 555517837 Sex: M Age: 46 DOB: 02/15/1963

DRG SERUM : Completed
DIG: 1.8

SIGNED BY LAWRENCE VON DOLLEN, MD (VON) 02/29/04

Patient Chart

Mccornack, Daniel

Date Printed: 09/02/09
555517837 Sex: M Age: 46 DOB: 02/15/1963

Laboratory Data

07/28/06 : 04:45pm
LAB RESULTS
Time collected: 0810
VON
Patient Name: McCornack, Dan
DOB: 02/15/63
Lab Performed: Central Coast Clinical Lab Temp
Data entered by: canderson
Date received: 01/25/07
Date entered: 01/25/07

Outside Ordering Physician: Gordon Lemm

LIP2: Completed
TG: 461***
T-CHOL: 232***
HDL: 36
CHOL/HDL: 6.4
HFP: Completed
BILI(T): 0.7
SGOT: 19
SGPT: 46
ALK PHOS: 62
ALB: 4.9
PROTEIN: 6.9
CMP: Completed
GLUCOSE: 88
NA: 140
K: 4.3
CL: 101
CO2: 21
ANION GAP: 22***
BUN: 25***
CR: 1.1
URIC A : 7.6***
CA: 10.0
CBC : Completed
WBC: 12.6***
RBC: 5.79
HGB: 17.7
HCT: 52.4***
MCV: 91
MCH: 30.6
MCHC: 33.8
PLAT CT: 158
DRG SERUM : Completed
DIGOXIN: 1.5

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Patient Chart

Mccornack, Daniel

Date Printed: 09/02/09

555517837 Sex: M Age: 46 DOB: 02/15/1963

PSAS: 0.55

SIGNED BY CHERYL ANDERSON (392) 01/25/2007 04:52PM

Printed using Practice Partner®

5-162

Patient Chart

Date Printed: 09/02/09

Mccornack, Daniel 555517837 Sex: M Age: 46 DOB: 02/15/1963

Laboratory Data

08/24/06: 05:24pm
LAB RESULTS
Time collected: 1134
VON
Patient Name: McCornack, Daniel
DOB: 02/15/63
Lab Performed: Central Coast Clinical Lab Temp
Data entered by: canderson
Date received: 01/23/07
Date entered: 01/23/07

Outside Ordering Physician: Gordon Lemm

LIP2: Completed
TSH: 1.896
----------Reviewed by: VON ----

SIGNED BY CHERYL ANDERSON (392) 01/23/2007 05:26PM

Patient Chart

Mccornack, Daniel

Date Printed: 09/02/09
555517837 Sex: M Age: 46 DOB: 02/15/1963

Laboratory Data

05/15/07 : 01:36pm
LAB RESULTS
Time collected: 0808
VON
Patient Name: McCornack, Daniel
DOB: 02/15/63
Lab Performed: Central Coast Clinical Lab Temp
Data entered by: canderson
Date received: 05/15/07
Date entered: 05/15/07

Outside Ordering Physician: Gordon Lemm

LIP2: Completed
TG: 620***
T-CHOL: 262***
HDL: 36
CHOL/HDL: 7.3***
HFP: Completed
BILI(T): 0.8
SGOT: 19
SGPT: 42
ALK PHOS: 62
ALB: 4.7
PROTEIN: 6.5
CMP: Completed
GLUCOSE: 106***
NA: 139
K: 4.6
CL: 101
CO2: 29
ANION GAP: 14
BUN: 23***
CR: 1.2
URIC A : 8.0***
CA: 9.7
ENDOCRINE : Completed
TSH: 3.670

DRG SERUM : Completed
DIGOXIN: 1.6

Printed using Practice Partner®

Patient Chart

Date Printed: 09/02/09

Mccornack, Daniel 555517837 Sex: M Age: 46 DOB: 02/15/1963

SIGNED BY LAWRENCE VONDOLLEN (VON) 05/16/2007 04:26PM

Patient Chart

Date Printed: 09/02/09

Mccornack, Daniel 555517837 Sex: M Age: 46 DOB: 02/15/1963

Laboratory Data

07/16/07 : 10:23am
LAB/PROTIME ONLY
PRO

Patient Name: MCCORNACK, DANIEL
DOB: 02/15/63

LAB TEST PROTIME
Time collected: 0822
Lab Performed: Central Coast Clinical Lab Temp
Data entered by: twolfe
Date received: 071607
Date entered: 071607

COUMADIN WE FOLLOW:COUMADIN WE FOLLOW
Trgt INR: 2.5-3.5
INR: 1.0
PT Sec: 12.2

SIGNED BY PROTIME NURSE (PRO) 07/17/2007 11:19AM

Printed using Practice Partner®

5-166

Patient Chart

Date Printed: 09/02/09

Mccornack, Daniel 555517837 Sex: M Age: 46 DOB: 02/15/1963

Laboratory Data

07/23/07 : 09:58am
LAB/PROTIME ONLY
PRO

Patient Name: MCCORNACK, DANIEL
DOB: 02/15/63

LAB TEST PROTIME
Time collected: 0805
Lab Performed: Central Coast Clinical Lab Temp
Data entered by: twolfe
Date received: 072407
Date entered: 072407

COUMADIN WE FOLLOW:COUMADIN WE FOLLOW
Trgt INR: 2.5-3.5
INR: 1.7
PT Sec: 18.0
DOSING: 5x7

SIGNED BY PROTIME NURSE (PRO) 07/25/2007 10:29AM

Printed using Practice Partner®

5-167

Patient Chart

Date Printed: 09/02/09

Mccornack, Daniel 555517837 Sex: M Age: 46 DOB: 02/15/1963

Laboratory Data

07/30/07 : 02:13pm
LAB/PROTIME ONLY
PRO

Patient Name: MCCORNACK, DANIEL
DOB: 02/15/63

LAB TEST PROTIME
Time collected: 0806
Lab Performed: Central Coast Clinical Lab Temp
Data entered by: twolfe
Date received: 073007
Date entered: 073007

COUMADIN WE FOLLOW:COUMADIN WE FOLLOW
Trgt INR: 2.5-3.5
INR: 2.8
PT Sec: 27.3
DOSING: 5x7

SIGNED BY PROTIME NURSE (PRO) 07/31/2007 09:29AM

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5-168

Cardiovascular Medicine and Cardiac Arrhythmias
An Incorporated Medical Group

Roger A. Winkle, M.D.
Edward T. Anderson, M.D.
R. Hardwin Mead, M.D.
Michael A. Ruder, M.D.
Nellis A. Smith, M.D.
Bruce A. Benedick, M.D.
Donald St. Claire, Jr, M.D.
Rob A. Patrawala, M.D.
Gregory Engel, M.D.

Diagnostic Cardiology
Cardiac Electrophysiology
Coronary Interventions
Cardiac Pacing
Nuclear Cardiology

1950 University Avenue, Suite 160 E. Palo Alto, CA 94303 650-617-8100 fax 650-327-2947
2900 Whipple Avenue, Suite 205 Redwood City, CA 94062 650-363-5262 fax 650-363-5265

JUNE 25, 2007

LAWRENCE VON DOLLEN, M.D.
295 POSADA LANE, SUITE A
TEMPLETON, CA 93465

GORDON LEMM, M.D.
292 POSADA LANE, SUITE D
TEMPLETON, CA 93465

RE: MCCORNACK, DANIEL
MR#: 58831

CARDIOLOGY CONSULTATION

CLINICAL HISTORY: This 44-year-old gentleman is referred for consideration of pulmonary vein ablation of atrial fibrillation. The patient had his first episode of probable atrial fibrillation at age 22. He would feel as if there were marbles or butterflies in his chest. He could get weak, cold, and tired. The episode lasted several days and he was treated with _____ caps. He was carried forward by, Dr. Harvey, who is now retired. Ultimately, Dr. Harvey told him he should stop his medications, as he was too young to be taking them. He stayed off of all medications for four years and had no major episodes. About four years later, he had another episode of sustained atrial fibrillation and went to his physician, who sent him immediately to the ER. That is when he first encountered Dr. Von Dollen. He was treated with digoxin in fairly high doses, which has now been cut back to 0.25-mg b.i.d. He was given Tenormin, which caused him to be a bit tired and fatigued and amiodarone, which caused him to have visual symptoms and actually possibly lose some vision. The patient subsequently has been treated with high dose diltiazem up to 480-mg daily in addition to 0.5-mg of digoxin. He thinks that he has some periods where he is in normal rhythm, but he is really not entirely certain as to whether he is fibrillating or not. When he lies down he is aware of an irregular beat. At times, he feels like he is in the normal rhythm and then develops an erratic rhythm. Before starting medications when he had atrial fibrillation he "felt like he would die." He does feel that he is tried and fatigued and not really a 100%. He notes no real precipitating factors for his bouts of atrial fibrillation. He was in atrial fibrillation for 24-hours on a Holter done by Dr. Von Dollen in June 2001. Several EKGs have shown him to be in atrial fibrillation. His left atrial size has been normal at 4.0-cm. He has no history of MIs, strokes, hypertension, thyroid disease, heart murmurs, rheumatic fever, asthma, or diabetes. He has occasional atypical chest pain. He has never had presyncope or syncope. He pops his head up with a pillow at night because he breaths better and has some heartburn. He has done this for at least several years or more. He has noted some mild edema over the last year. His cholesterols have been elevated. One

5-170

TO: LAWRENCE VON DOLLEN, M.D.
GORDON LEMM, M.D.
R MCCORNACK, DANIEL
DATE: 06/25/2007
Page 2

done in May 2007 was 262 with HDL of 36 and triglycerides of 620, so the LDL could not be determined. A TSH at the time was 3.67.

PAST MEDICAL HISTORY:

OPERATIONS: He had a tonsillectomy at age 19. He had left knee arthroscopic surgery in 1993.

MEDICATIONS:
1. Diltiazem 300-mg and 180-mg daily.
2. Allopurinol 100-mg t.i.d.
3. Digitek 0.25-mg b.i.d.
4. Prevacid 30-mg daily.
5. Aspirin 325-mg daily.

ALLERGIES: Ampicillin cause some swelling, sulfa drugs cause swelling and redness, atenolol cause fatigue, and amiodarone cause some visual decline.

FAMILY HISTORY: All of his grandparents are alive. His mother is alive at 62 and father at 64 in good health. He has a brother, 37 in good health. He has a sister, 41 in good health. He has a son 14 and a son 16 both in good health.

SOCIAL HISTORY: He uses Copenhagen chewing tobacco, quitting four months ago. He has two to [illegible] beers daily. He has three cups of coffee daily. He is married. He is a plant manager for a custom [illegible]emical manufacturing plant. He is married and his wife comes with him today.

REVIEW OF SYSTEMS: He had some fevers and sweats and underwent an evaluation and was found to have an HLA 27 A-gene. He has some minor back issues, which sound more like disc disease, and ankylosing spondylitis. He had a past history of possible ulcers.

PHYSICAL EXAMINATION:
GENERAL: He is a mildly overweight middle-aged gentleman and in no distress.
VITAL SIGNS: Blood pressure is 140/85. Weight is 223.
HEENT: Negative.
NECK: No JVD. No carotid bruits. Thyroid not enlarged.
LUNGS: Clear.
HEART: Rhythm irregularly irregular at about 70 to 80 per minute. No clicks, murmurs, gallops, or rubs.
ABDOMEN: Soft. Bowel sounds active. No organomegaly. No bruits.
EXTREMITIES: No edema. Pedal pulses 1+ and equal.

IMPRESSION:
1. Atrial fibrillation. This clearly was paroxysmal in the past. It is very difficult to tell if it is persistent or permanent at this time. He has been in atrial fibrillation most of the time Dr. Von Dollen has seen him. The patient thinks that he has periods where he is in normal rhythm and then goes into atrial fibrillation. He has a lot of fatigue and lack of energy, which he attributes to his atrial fibrillation. It is really not clear if he is having periods of sinus rhythm to compare the atrial fibrillation symptoms to. I asked him to wear an iCardia monitor for a month and send us

5-171

TO: LAWRENCE VON DOLLEN, M.D.
GORDON LEMM, M.D.
R)CCORNACK, DANIEL
DA:E: 06/25/2007
Page 3

strips frequently especially when he thinks he might be in normal rhythm. If in fact he is still having periods of sinus rhythm it might be worth a trial of an antiarrhythmic drug such as propafenone or flecainide to see if he feels better and sustain sinus rhythm. If he is in permanent atrial fibrillation it might be worth doing a cardioversion on propafenone or flecainide to see if we could get a few weeks of sinus rhythm to see if he felt better. I explained to him the invasive nature of a pulmonary vein isolation procedure. He understands the need for transseptal puncture and risk of 1% including death, stroke, perforation, atrial esophageal fistulae, pulmonary vein stenosis, groin complications and other serious complications. He understands there is a 30% need for a second procedure. If he is in permanent atrial fibrillation his cure rate would be 60% to 65% and if he is in paroxysmal atrial fibrillation it would be 70% to 75%. I explained that the only known benefit was improvement in quality of life. Although, there are theoretical reasons that think sinus rhythm would decrease the risk of stroke and other complications. There is no scientific prove in this regard. After he sends the strips to us we will decide whether or not to try to restore sinus rhythm briefly to see if he feels dramatically better. If so he certainly would be an excellent candidate for ablation. If he is highly motivated to restore sinus rhythm. He may just want to proceed directly to a pulmonary vein isolation procedure to try to get off of all of his current drugs.

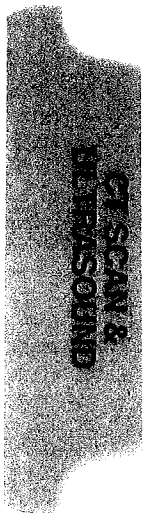
2. HLA 27 gene positive with relatively few symptoms related to this.
3. Mild edema for the past year. This certainly could be aggravated by his diltiazem. He has been on it for a long time and it is possible that this is when he went into permanent atrial fibrillation.

)ER A. WINKLE, M.D.
Dictated, but not read or signed.

RW/PSG/PSG
DD: 06/25/2007
DT: 06/28/2007
FILENAME: 07062501-RWINKLE-062507-MCCORNACK-DANIEL-58831

DEMCM:0056

5-172


CT SCAN &
ULTRASOUND

Patient Chart

Mccornack, Daniel 555517837 Sex: M Age: 46 DOB: 02/15/1963

Date Printed: 09/02/09

Echo/CardiacMR

02/23/95
Echocardiogram/ Twin Cities Community Hospital
Echo/ TCCH*
Patient Name: MCCORNACK, DANIEL
ECHO: completed

ECHOCARDIOGRAPHY REPORT
McCornack, Daniel
02/23/95

1. The right atrium, right ventricle, tricuspid and pulmonicvalves are grossly normal.
2. The left atrial size is normal. No intracavitary masses areseen.

3. The mitral valve leaflets move well without evidence of stenosis, thickening, prolapse, systolic anterior motion, vegetations or masses.

4. The left ventricular internal diastolic dimension is normal. The interventricular septum and left ventricular posterior free wall are normal thickness. The overall chamber size, wall thickness, wall motion and ejection fraction are well within the mid-range of normal.

5. The aortic root diameter is normal. The valve has three leaflets which move normally without significant stenosis.

6. No significant pericardial effusion is seen.

7. No significant valvular stenosis is seen as evidenced by normal peak flow velocity of the mitral, tricuspid, pulmonic and aortic valves. No significant valvular regurgitation is seen.

8. Color flow doppler shows no significant mitral, tricuspid, aortic or pulmonic stenosis or regurgitation.

IMPRESSION
1. GROSSLY NORMAL M-MODE AND TWO-DIMENSIONAL STUDY.

D: 03/05/95
T: 03/06/95
LVD:pk
Lawrence Von Dollen, M.D.

SIGNED BY LAWRENCE VON DOLLEN, MD (VON) 07/31/01

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5-174

Patient Chart

Date Printed: 09/02/09

Mccornack, Daniel 555517837 Sex: M Age: 46 DOB: 02/15/1963

Echo/CardiacMR

06/12/01
COASTAL CARDIOLOGY NON-INVASIVE LABORATORY
ECHOCARDIOGRAM REPORT
ECHO:completed

Coastal Cardiology
77 Casa Street, Suite 104
San Luis Obispo, California 93405
(805) 782-8844 - FAX (805) 782-8850

Patient Name: MCCORNACK, DANIEL
Referring Physician: Gordon Lemm, M.D.
Cardiologist: Lawrence Von Dollen, M.D., F.A.C.C.
Technician: Katy Phillips, RDCS, RVT

Ht: 72 Wt: 200 Tape: 116/01 Footage#: 27-33

Clinical Complaint: Irregular heart beat
Clinical Diagnosis: Atrial fibrillation

ECHOCARDIOGRAPHIC DATA-MEASUREMENTS
Left Atrium-End Systole (Normal 2.5-4.4 cm): 4.1
Right Ventricle-End Diastole (Normal <3.0 cm): 2.0
Aortic Root Diameter (Normal 2.0-4.0 cm): 2.8
Aortic Cusp Excursion (Normal 1.5-2.0 cm): 1.9
E-Point to Septal Separation (Normal </= 1.0 cm): 0.7
Interventricular Septum-End Diastole (Normal 0.3-0.8 cm): 1.0
Interventricular Septum-End Systole (Normal 0.6-1.6 cm): 1.5
Left Ventricular Posterior Wall-End Diastole (Normal 0.5-1.3 cm): 1.1
Left Ventricular Posterior Wall-End Systole (Normal 0.9-1.4 cm): 1.5
Left Ventricle-End Diastole (Normals <5.8 cm): 4.9
Left Ventricle-End Systole: 3.7
Left Ventricular Fractional Shortening (Normal >24%): 24%
Left Ventricular Ejection Fraction (rest)(Normal >55%): 50%

2D MEASUREMENTS:

DOPPLER MEASUREMENTS:
-Aortic Valve-
Left Ventricular Outflow Tract Velocity (V1): 0.71 m/s
Peak Aortic Velocity: 1.0 m/s
Aortic Regurgitation Severity: none seen
-Mitral Valve-
Peak Velocity (E)(Normal 0.6-1.0 m/s): 0.96 m/s
Mitral Regurgitation Severity: trace
-Tricuspid Valve-
Peak Velocity (systole): 1.1 m/s

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Patient Chart

Date Printed: 09/02/09

Mccornack, Daniel 555517837 Sex: M Age: 46 DOB: 02/15/1963

Right Atrial Pressure: 10 mmhg
Right Ventricular Pulmonary Artery Systolic Pressure: 14.8 mmHg
Tricuspid Regurgitation Severity: whiff
-Pulmonic Valve-
Pulmonic Regurgitation Severity: whiff

INTERPRETATION: Grossly normal echocardiographic study with normal left ventricular wall motion and ejection fraction of 70%. Clinically insignificant mitral, tricuspid and pulmonic insufficiency is seen.

Lawrence Von Dollen, M.D., F.A.C.C./lt D: 06/15/01 T: 06/20/01

SIGNED BY LAWRENCE VON DOLLEN, MD (VON) 06/20/01

Printed using Practice Partner®

5-176

Patient Chart

Date Printed: 09/02/09

Mccornack, Daniel 555517837 Sex: M Age: 46 DOB: 02/15/1963

Echo/CardiacMR

12/14/06 : 01:51pm
COASTAL CARDIOLOGY NON-INVASIVE LABORATORY
ECHOCARDIOGRAM REPORT
ECHO:completed
VON
Patient Name: MCCORNACK, DANIEL
Date of Birth: 02/15/63
Referring Physician: Gordon Lemm, M.D.
Cardiologist: Lawrence Von Dollen, M.D., F.A.C.C.
Technician: JKovacs, RVT

Ht: Wt: Tape: T110 Footage#: 8:00

Clinical Complaint:
Clinical Diagnosis: A-Fib

ECHOCARDIOGRAPHIC DATA-MEASUREMENTS
Left Atrium-End Systole (Normal 2.5-4.4 cm): 4.0
Right Ventricle-End Diastole (Normal <3.0 cm): 2.0
Aortic Root Diameter (Normal 2.0-4.0 cm): 3.2
Aortic Cusp Excursion (Normal 1.5-2.0 cm): 1.9
E-Point to Septal Separation (Normal </= 1.0 cm): .5
Interventricular Septum-End Diastole (Normal 0.3-0.8 cm): .9
Interventricular Septum-End Systole (Normal 0.6-1.6 cm): 1.3
Left Ventricular Posterior Wall-End Diastole (Normal 0.5-1.3 cm): .6
Left Ventricular Posterior Wall-End Systole (Normal 0.9-1.4 cm): .9
Left Ventricle-End Diastole (Normals <5.8 cm): 5.6 Left Ventricle-End Systole: 3.9
Left Ventricular Fractional Shortening (Normal >24%): 30
Left Ventricular Ejection Fraction (rest)(Normal >55%): 57

DOPPLER/COLOR MEASUREMENTS:
-Aortic Valve-
Left Ventricular Outflow Tract Velocity (V1): .71
Peak Aortic Velocity: 1.3
Left Ventricular Outflow Tract Diameter: 2.2
Aortic Valve Area: 3.0
Aortic Valve Gradient (peak): 6.8
Aortic Regurgitation Severity: None Seen
-Mitral Valve-
Peak Velocity (E)(Normal 0.6-1.0 m/s): .87
Deceleration Time (E-wave)(160-230msec): 250
Mitral Valve Area: 3.0
Mitral Valve Gradient (peak): 3.0
Mitral Regurgitation Severity: Trace
-Tricuspid Valve-
Right Atrial Pressure: 10
Tricuspid Regurgitation Severity: None Seen
-Pulmonic Valve-
Pulmonic Regurgitation Severity: None Seen

INTERPRETATION:
1. The right atrium, right ventricle, tricuspid and pulmonic valves are normal. The RVIDD is normal.

Printed using Practice Partner®

Patient Chart

Mccornack, Daniel

Date Printed: 09/02/09

555517837 Sex: M Age: 46 DOB: 02/15/1963

2. The left atrial size of 40mm is normal. There are no intracavitary masses or thrombi noted. The intra atrial septum is grossly normal without obvious defects, masses, or aneurism.
3. The mitral valve leaflets are normal without significant thickening, stenosis, prolapse, SAM, vegetations, or masses.
4. The left ventricular chamber size, wall thickness, wall motion and estimated ejection fraction of 75 % are normal.
5. The aortic root diameter is normal. The aortic valve is normal with three leaflets without significant thickening and with normal motion.
6. No significant pericardial effusion is noted.
7. There is no significant stenosis of the tricuspid, pulmonic, mitral, or aortic valves. There is no significant regurgitation of the tricuspid, pulmonic, mitral or aortic valves.
Clinically very mild mitral and tricuspid regurgitation are present.

CONCLUSION: Essentially normal M-Mode, two dimensional, and Doppler echocardiographic study.

SIGNED BY LAWRENCE VON DOLLEN (VON) 12/18/2006 08:04AM

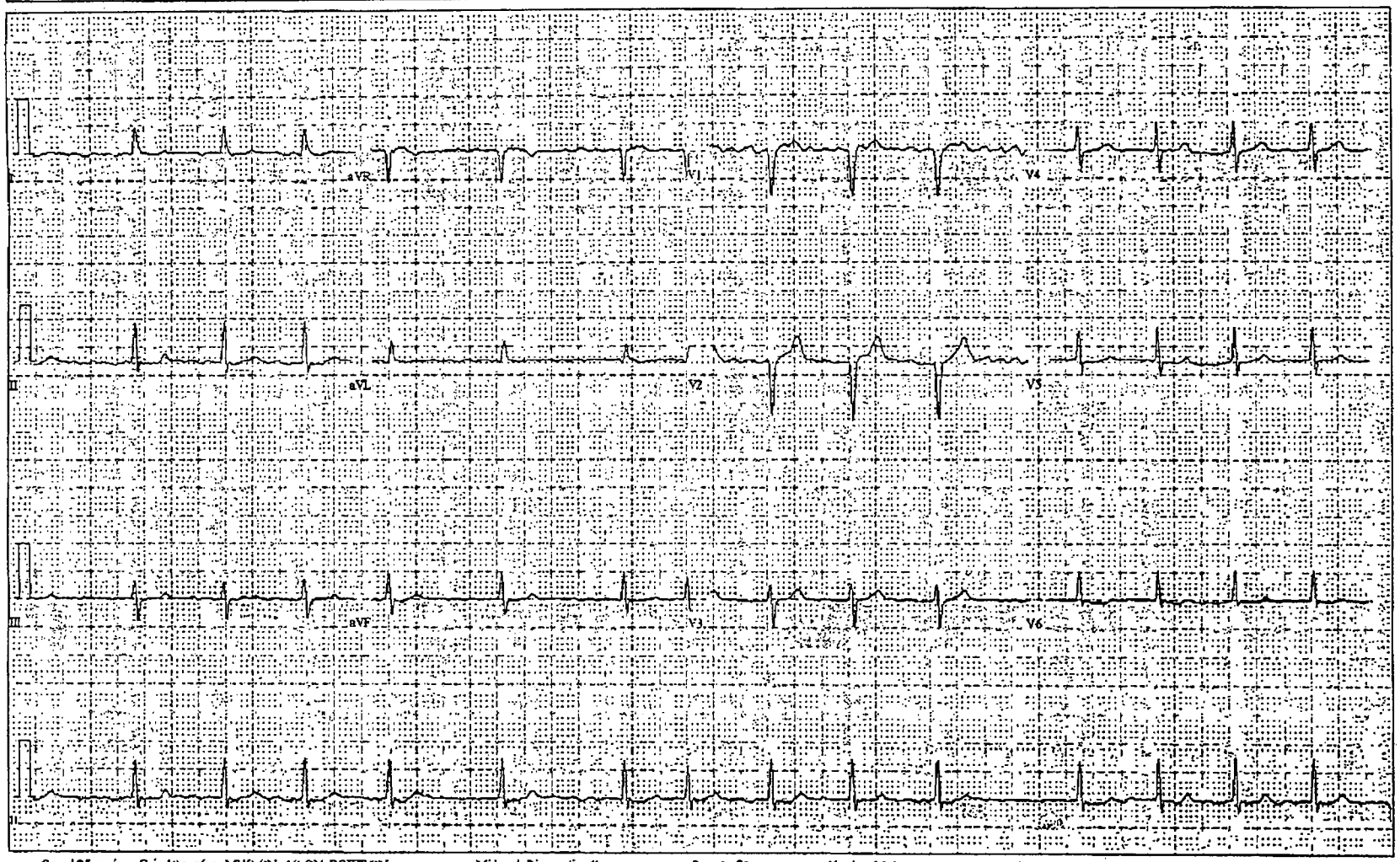
EKG

5-179

5-180

Coastal Cardiology

Name:	Daniel McCormack	Req. Physician:		Rate:	89	BPM	Interpretation:
ID:	555517837	Technician:	CMartin/Vondollen	PR:	-	msec	Atrial fibrillation -irregular conduction
Sex:	Male	History:		QT/QTc:	324/376	msec	- Nonspecific ST-T-abnormality .
BP:		Medication:		QRSD:	98	msec	
Weight:	lbs	Date of Report:	02/01/00 10:00:36	P Axis:	-		ABNORMAL
Height:	inches	Reviewed By:	lvondollen	QRS Axis:	26		
Age:	36 Years	Review Date:	06/17/00 14:14:44	T Axis:	57		
Comments:							



Speed:25 mm/sec Gain:10 mm/mv MYO:ON AC:ON DRIFT:ON Midmark Diagnostics Group Page 1 of 1 Version 6.3.0 Print Date 09/02/09 14:37:58

Coastal Cardiology

Name:	Daniel McCormack	Req. Physician:		Rate:	96	BPM	Interpretation:
ID:	555517837	Technician:	MMontogmery/VanDollen	PR:	-	msec	Atrial fibrillation
Sex:	Male	History:		QT/QTc:	332/396	msec	- Nonspecific ST-T abnormality
BP:		Medication:		QRSD:	104	msec	
Weight:	lbs	Date of Report:	12/18/00	P Axis:	-		ABNORMAL
Height:	inches	Reviewed By:	Ivandollen	QRS Axis:	4		
Age:	37 Years	Review Date:	03/17/01 15:41:04	T Axis:	-		

Comments:

Speed25 mm/sec Gain10 mm/mv MYOION ACSON DRIFTON Midmark Diagnostics Group Page 1 of 1 Version 6.3.0 Print Date 09/02/09 14:37:54

5-181

AmbECG/Amb BP/Event Rec

Date Printed: 09/02/09

Name: Mccornack, Daniel ID: 555517837 SEX:M AGE:46

06/12/01
COASTAL CARDIOLOGY NON-INVASIVE LABORATORY
AMBULATORY ECG INTERPRETATION
HOLTER . completed

Patient Name: MCCORNACK, DANIEL
Cardiologist: Lawrence Von Dollen, M.D.

Referring Physician: Gordon Lemm, M.D.
Technician: D. Houston
Date Received: 06/14/01
Date Scanned: 06/19/01

Clinical Complaint: Atrial Fibrillation

Medication: Lanoxin, Dilacor
Pacemaker: No

ECTOPIC SUMMARY:
The patient was monitored for a period of: 20 hours and 00 minutes
The total number of beats was: 118436
The average heart rate was: 105
The maximum heart rate was: 185
The minimum heart rate was: 71
Wide beats totaled: 19
Wide couplets totaled: 0
Wide runs totaled: 0
Pauses totaled: 0
Narrow runs totaled: 0
Isolated early narrow beats totaled: 0

ISCHEMIC SUMMARY:
ST segment depression: Maximum ST inaccurate due to artifact.

CONCLUSION: Chronic atrial fibrillation
Atrial fibrillation with slightly increased average ventricular response. No significant pauses or asystolic or tachycardic spells were noted. Symptoms of irregular rapid heart beat showed no basic change in the rate or rhythm.

SIGNED BY LAWRENCE VON DOLLEN, MD (VON) 07/05/01

PLAINTIFFS' EXHIBITS 012533

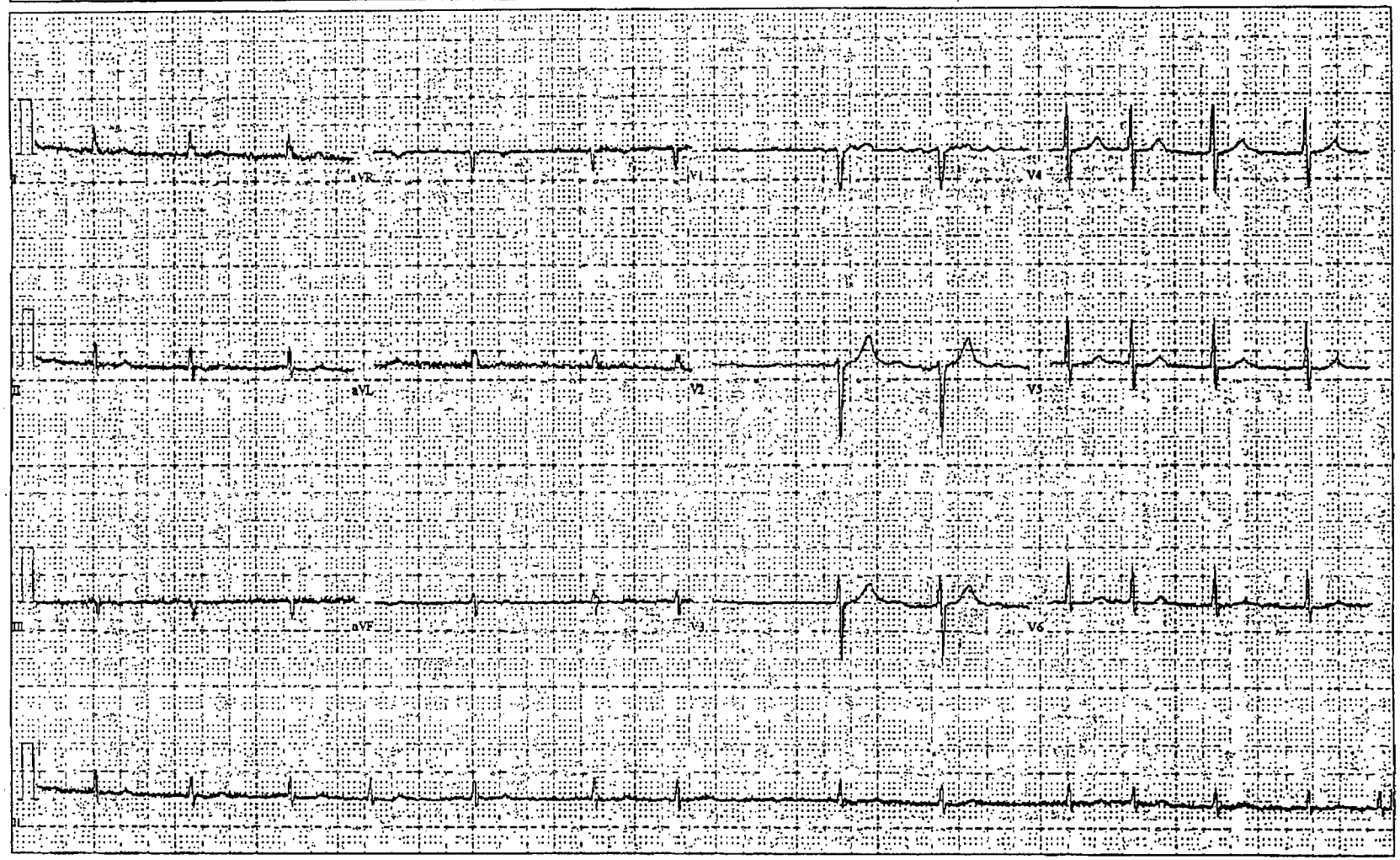
5-183

Name:	Daniel McCormack	Coastal Cardiology		Rate:	81	BPM	Interpretation:
ID:	555517837	Req. Physician:	Lawrence Von Dollen, M.D.	PR:	-	msec	Atrial fibrillation
Sex:	Male	Technician:	Yolanda Leppelman, MA	QT/QTc:	320/357	msec	- Nonspecific T-abnormality .
BP:		History:		QRSD:	88	msec	
Weight	lbs	Medication:		P Axis:	-		ABNORMAL
Height:	inches	Date of Report:	11/27/06 11:43:46	QRS Axis:	-9		
Age	43 Years	Reviewed By:	Lawrence Von Dollen, M.D.	T Axis:	-1		
Comments:		Review Date:	11/27/06 11:54:31				

Speed:25 mm/sec Gain:10 mm/mv MYO:ON AC:ON DRIFT:ON Midmark Diagnostics Group Page 1 of 1 Version 6.3.0 ECG Analysis Ver. 6.3.0 Print Date 09/02/09 14:37:46

Coastal Cardiology

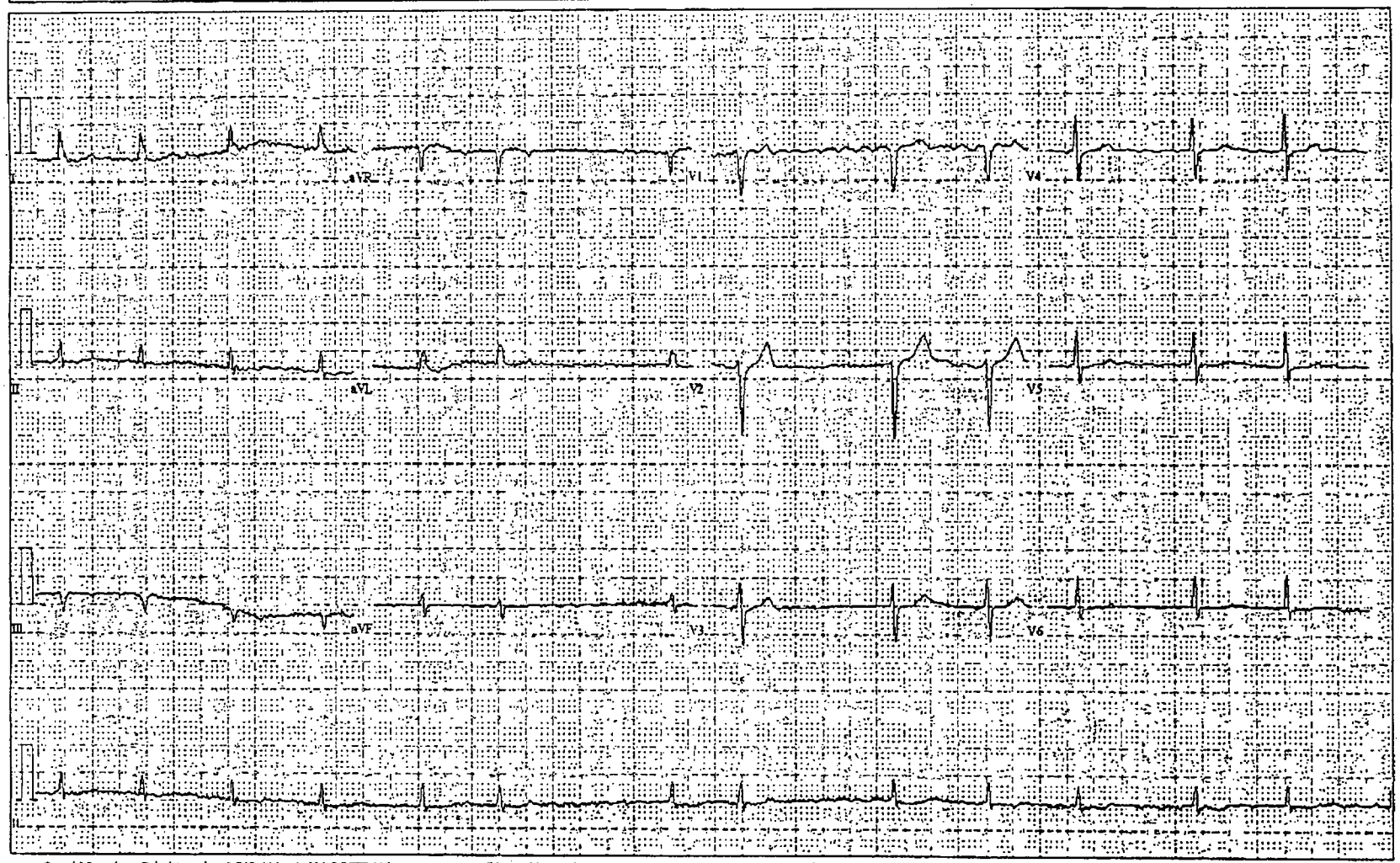
Name:	Daniel McCornack	Req. Physician:	Lawrence Von Dollen, M.D.	Rate:	82	BPM	Interpretation:
ID:	555517837	Technician:	Peggy Callison, RN	PR:	-	msec	Atrial fibrillation -irregular conduction
Sex:	Male	History:		QT/QTc:	326/365	msec	Conduction ratio >= 2, low amplitude P waves,
BP:		Medication:		QRSD:	82	msec	irregular RR
Weight:	lbs	Date of Report:	07/13/07 11:49:48	P Axis:	-		ABNORMAL RHYTHM
Height:	inches	Reviewed By:	Lawrence Von Dollen, M.D.	QRS Axis:	-12		
Age:	44 Years	Review Date:	07/13/07 23:36:06	T Axis:	-1		
Comments:							



Speed:25 mm/sec Gain:10 mm/mv MYO:OFF AC:ON DRIFT:ON Midmark Diagnostics Group Page 1 of 1 Version 6.3.0 ECG Analysis Ver. 6.3.0 Print Date 09/02/09 16:37:38

Coastal Cardiology

Name:	Daniel McCormack	Req. Physician:	Lawrence Von Dollen, M.D.	Rate:	79	BPM	Interpretation:
ID:	555517837	Technician:	Sakisha Alexander	PR:	-	msec	Atrial flutter-fibrillation
Sex:	Male	History:		QT/QTc:	324/358	msec	Conduction ratio >= 2, med amplitude P waves, no
BP:		Medication:		QRSD:	88	msec	atrial rhythm
Weight:	lbs	Date of Report:	11/29/07 15:20:06	P Axis:	-		- Nonspecific T-abnormality
Height:	inches	Reviewed By:	Lawrence Von Dollen, M.D.	QRS Axis:	-11		
Age:	44 Years	Review Date:	12/03/07 14:05:31	T Axis:	-1		ABNORMAL
Comments:							



Speed:25 mm/sec Gain:10 mm/mv MYO:ON AC:ON DRIFT:ON Midmark Diagnostics Group Page 1 of 1 Version 6.3.0 ECG Analysis Ver. 6.3.0 Print Date 09/02/09 14:36:53

CERTIFICATE OF DEATH &
AUTOPSY (ORIGINAL)

STATE OF CALIFORNIA
CERTIFICATION OF VITAL RECORDS

COUNTY OF SANTA CRUZ

SANTA CRUZ, CALIFORNIA

CERTIFICATE OF DEATH

STATE OF CALIFORNIA — USE BLACK INK ONLY / NO ERASURES, WHITEOUTS OR ALTERATIONS

STATE FILE NUMBER: 3200844000402

LOCAL REGISTRATION NUMBER:

DECEDENT'S PERSONAL DATA

1. NAME OF DECEDENT — FIRST (Given): DANIEL
2. MIDDLE: ELWIN
3. LAST (Family): MCCORNACK
AKA: ALSO KNOWN AS — (Include AKA (FIRST, MIDDLE, LAST))
4. DATE OF BIRTH mm/dd/ccyy: 02/15/1963
5. AGE-Yrs: 45
If UNDER ONE YEAR — Months / Days
If UNDER 24 HOURS — Hours / Minutes
6. SEX: M
7. BIRTH STATE/FOREIGN COUNTRY: CA
8. SOCIAL SECURITY NUMBER: 555-51-7837
9. EVER IN U.S. ARMED FORCES? YES / NO / UNK
10. MARITAL STATUS (At Time of Death): MARRIED
11. DATE OF DEATH mm/dd/ccyy: 03/23/2008
12. HOUR (24 Hour): 0052
13. EDUCATION (Highest Degree — see instructions): HS GRADUATE
14. WAS DECEDENT HISPANIC/LATINO? YES / NO / UNK
15. DECEDENT'S RACE — Up to 6 races may be listed (see instructions on back): CAUCASIAN
16. USUAL OCCUPATION — Type of work for most of life, DO NOT use RETIRED: PLANT MANAGER
17. KIND OF BUSINESS/INDUSTRY: CHEMICAL MANUFACTURE
18. YEARS IN OCCUPATION: 25

USUAL RESIDENCE

20. DECEDENT'S RESIDENCE (Street and number or location): 6255 PEACHY CANYON RD.
21. CITY: PASO ROBLES
22. COUNTY/PROVINCE: SAN LUIS OBISPO
23. ZIP CODE: 93446
24. YEARS IN COUNTY: 45
25. STATE/FOREIGN COUNTRY: CA

INFORMANT

26. INFORMANT'S NAME, RELATIONSHIP: KATHY MCCORNACK, WIFE
27. INFORMANT'S MAILING ADDRESS (Street and number or rural route number, city or town, state, ZIP): 6255 PEACHY CANYON RD, PASO ROBLES, CA 93446

SPOUSE / PARENT INFORMATION

28. NAME OF SURVIVING SPOUSE — FIRST: KATHY
29. MIDDLE: MARIE
30. LAST (Married Name): ESPARZA
31. NAME OF FATHER — FIRST: RALPH
32. MIDDLE: MICHAEL
33. LAST: MCCORNACK
34. BIRTH STATE: CA
35. NAME OF MOTHER — FIRST: LINDA
36. MIDDLE: EILEEN
37. LAST (Maiden): HIRSCHLER
38. BIRTH STATE: CA

FUNERAL DIRECTOR / LOCAL REGISTRAR

39. DISPOSITION DATE mm/dd/ccyy: 03/28/2008
40. NAME OF FINAL DISPOSITION: PASO ROBLES DISTRICT CEMETERY
41. PLACE OF DISPOSITION(S): BU
42. STORAGE FACILITY: UNDETERMINED
43. LICENSE NUMBER
44. NAME OF FUNERAL ESTABLISHMENT: KUEHL-NICOLAY FUNERALS AND CREM
45. LICENSE NUMBER: FD68
46. REGISTRAR: P. PORI NAMKUNG, M.D.
47. DATE mm/dd/ccyy: 03/27/2008

PLACE OF DEATH

48. PLACE OF DEATH: CAMPSITE
49. COUNTY: SANTA CRUZ
50. FACILITY ADDRESS OR LOCATION (Street and number or rural route number): 4779 SITE 1 HIGHWAY 9
51. CITY: FELTON

CAUSE OF DEATH

52. CAUSE OF DEATH — Enter only one cause per line for (a), (b), (c), and (d). DO NOT abbreviate.
IMMEDIATE CAUSE (Final disease or condition resulting in death): (a) CARDIAC ARREST
Sequentially list conditions, if any, leading to cause on line a. Enter UNDERLYING CAUSE (disease or injury that initiated events resulting in death) LAST: (b) VENTRICULAR ARRHYTHMIA
(c) ATRIAL FIBRILLATION
(d) HYPERTENSIVE AND ARTERIOSCLEROTIC CARDIOVASCULAR DISEASE
53. OTHER SIGNIFICANT CONDITIONS CONTRIBUTING TO DEATH BUT NOT RESULTING IN THE UNDERLYING CAUSE GIVEN IN 52: EXOGENOUS OBESITY
54. WAS OPERATION PERFORMED FOR ANY CONDITION IN ITEM 52 OR 53? NO
55. IF FEMALE, PREGNANT IN LAST YEAR? YES / NO / UNK
56. IDENTIFY THAT TO THE BEST OF MY KNOWLEDGE DEATH OCCURRED AT THE HOUR, DATE, AND PLACE STATED FROM THE CAUSES STATED. Decedent Attended Since: (a) mm/dd/ccyy; Decedent Last Seen Alive: (b) mm/dd/ccyy
57. SIGNATURE AND TITLE OF CERTIFIER
58. LICENSE NUMBER
59. DATE mm/dd/ccyy
60. TYPE/PRINT ATTENDING PHYSICIAN'S NAME, MAILING ADDRESS, ZIP CODE
61. I CERTIFY THAT IN MY OPINION DEATH OCCURRED AT THE HOUR, DATE, AND PLACE STATED FROM THE CAUSES STATED. MANNER OF DEATH: Natural / Accident / Homicide / Suicide / Pending Investigation / Could not be determined
62. INJURED AT WORK? YES / NO / UNK
63. INJURY DATE mm/dd/ccyy
64. HOUR (24 Hour)
65. PLACE OF INJURY (e.g., home, construction site, wooded area — city)
66. DESCRIBE HOW INJURY OCCURRED (Events which resulted in injury)
67. LOCATION OF INJURY (Street and number or location, and city and ZIP)

CORONER'S USE ONLY

68. SIGNATURE OF CORONER / DEPUTY CORONER: NAOMI SILVA
69. DATE mm/dd/ccyy: 03/27/2008
70. TYPE/PRINT NAME, TITLE OF CORONER / DEPUTY CORONER: NAOMI SILVA, DEPUTY CORONER

STATE REGISTRAR: a. b. c. d. e.
012008000773428
FAX AUTH. #
CENSUS TRACT

CERTIFIED COPY OF VITAL RECORDS
STATE OF CALIFORNIA
COUNTY OF SANTA CRUZ
DATE ISSUED APR 07 2008
00018937O

This is a true and exact reproduction of the document officially registered and placed on file in the Vital Records Section, Santa Cruz County Public Health Department.

Pori Namkung
CHIEF PUBLIC HEALTH OFFICER
SANTA CRUZ, CALIFORNIA

This copy not valid unless prepared on engraved border displaying seal and signature of Registrar.

SANTA CRUZ COUNTY SHERIFF-CORONER'S OFFICE
701 Ocean Street
Santa Cruz, California
* REPORT OF AUTOPSY EXAMINATION *

AUTOPSY NUMBER: CA08-037 **FILE NUMBER:** 08-02790

NAME: Daniel Mc Cornack **AGE:** 45 **SEX:** Male

PLACE OF DEATH: Smithwood R.V. Park, 4770 Hwy 9, Felton

DATE/HOUR OF DEATH: March 23, 2008 @ 0052 Hours

AUTOPSY PERFORMED: Santa Cruz County Morgue

DATE/HOUR OF AUTOPSY: March 26, 2008 @ 7:30 a.m.

PATHOLOGIST: Richard T. Mason, M.D.

BODY IDENTIFIED BY: Ankle tag.

ATTENDING PHYSICIAN: None.

CAUSE OF DEATH: CARDIAC ARREST
Due to: Ventricular arrhythmia
Due to: Atrial fibrillation
Due to: Hypertensive and arteriosclerotic cardiovascular disease.

CONTRIBUTORY: Exogenous obesity.

MANNER: Natural.

DIAGNOSES:

1. Hypertensive and arteriosclerotic cardiovascular disease with:

EXHIBIT 2
Deponent Mason
Date 10/1/09 Rptr. AAH

PLT DEM,Sr.(2)00010

DIAGNOSES, continued

A. Cardiomegaly and left ventricular hypertrophy.
B. Coronary arteriosclerosis, mild to moderate.
C. Myocardial fibrosis, mild.
D. Atrial fibrillation by history.
E. Probable ventricular arrhythmia and arrest.

2. Cerebral edema and congestion.

3. Pulmonary edema and congestion.

4. Exogenous obesity, moderate.

RICHARD T. MASON, M.D.
Forensic Pathologist

RTM/dp

EXTERNAL EXAMINATION

The body examined is that of a well-developed, mildly obese, middle-aged white male that appears the stated age of 45 years. The body is 70 inches in length and weighs 220 pounds. The scalp hair is medium brown with gray and is cut short measuring 1/4 inch. The eyes are blue gray with the pupils equal in diameter, measuring 6 mm. There is an adhesive nostril dilating device attached over the midportion of the nostrils. There is a short 3/4 inch grayish brown mustache. Natural teeth in good condition are present in the mouth. There is a 1-2 mm growth of beard present on the lower face. There is prominent pinkish cyanosis of the anterior face and neck.

Examination of the anterior chest reveals 4 x 6 inch adhesive defibrillator electrodes present over the left lower lateral chest and the right upper anterior chest. Adhesive EKG electrodes are present over the right and left upper anterior chest and the right and left lower abdomen. The axillae are normal.

Examination of the anterior abdomen reveals it to be mildly obese. There is a slight umbilical hernia. There are no other marks or wounds are noted on the anterior abdomen. Normal male external genitalia are present. The penis is circumcised.

Examination of the lower limbs reveals normal, symmetric, muscular right and left thighs and right and left lower legs. There is a coroner's identification band present on the right ankle bearing the name: McCornack, Daniel; #08-2790. The right and left feet are normal.

Examination of the upper limbs reveals normal, symmetric, muscular right and left upper arms and right and left forearms.

The antecubital spaces are clean with no marks or wounds. The right and left forearms are unremarkable. An intravenous line is in position through a needle puncture wound on the dorsum of the left hand. This line is attached to a 1-liter bag of normal saline.

Examination of the hands reveals them to be normal with short intact fingernails.

INTERNAL EXAMINATION

HEAD:
Reflection of the scalp reveals an absence of any contusions on the galeal surface. The calvarium is intact. Reflection of the calvarium reveals prominent cerebral edema. The gyri are flattened. The meninges are clear but congested. The brain weighs 1,640 grams. The brain has a normal external morphology except for the edema. The cerebral arteries are normal in distribution and appearance.

Multiple coronal sections through both cerebral hemispheres reveal normal cortex, normal white matter and normal basal ganglia. Sections through the brainstem and cerebellum reveal these structures to be normal.

The dura is stripped from the base of the skull to reveal an intact skull base.

NECK:
The hyoid bone, larynx, trachea, soft tissues, cervical spine are intact. The airway is fully patent.

BODY CAVITIES:
The pericardial cavity contains 25 mL of clear yellow fluid. There is no excess fluid in the pleural or peritoneal cavities.

CARDIOVASCULAR SYSTEM:
Heart weight 500 grams. There is cardiomegaly and left ventricular hypertrophy. The epicardial surfaces are smooth and glistening. The heart valves are normal. The atria are normal in size. The endocardial surfaces of the atria and ventricles are normal in appearance. Dissection of the coronary arteries reveals abundant, scattered, flattened atherosclerotic plaque in the right coronary artery, which is of greatest circumference compared to the LAD and the circumflex coronary arteries. There is flattened atherosclerotic plaque in a small left anterior descending coronary artery. There is a minimal amount of atherosclerotic plaque in the left circumflex coronary artery. Multiple cross sections through both ventricles of the heart reveal some mild diffusely distributed myocardial fibrosis. There is cardiomegaly and left ventricular hypertrophy with the left ventricle measuring 16 mm in thickness and the right ventricle measuring 4 mm in thickness. There are no foci or evidence of old or recent myocardial infarction.

Examination of the aorta reveals it to be smooth with minimal focal atherosclerosis. The superior and inferior vena cavae are intact and normal with no thromboemboli.

RESPIRATORY TRACT:
Lungs, weight right 830 grams, left 840 grams. There is severe bilateral pulmonary edema and congestion. Bloodstained watery fluid runs from the cut surfaces of all lobes of both lungs. There are no foci of consolidation. The major bronchi contain a small amount of bloodstained edema fluid. The pulmonary arteries are widely patent with no thromboemboli.

LIVER:
Weight 2,550 grams. The smooth, light, reddish tan capsular surface is intact. The liver is enlarged and there is fatty metamorphosis. The parenchyma is light pinkish tan and fractures easily on digital pressure. There is no increase in fibrous tissues to palpation. The intra and extrahepatic blood vessels and bile ducts are grossly normal. The gallbladder is thin-walled and contains 1 mL of light brown transparent bile.

SPLEEN:
Weight 470 grams. This organ is enlarged and congested. The dark gray brown capsular surface is intact with no evidence of trauma. The parenchyma is dark red brown firm.

PANCREAS:
Weight 210 grams. Normal, pale tan, lobular, autolyzed parenchyma is noted on cut section.

ENDOCRINE SYSTEM:
The pituitary, adrenal and thyroid glands are grossly normal.

GENITOURINARY TRACT:
Kidneys, weight right 230 grams, left 220 grams. The cortical surfaces of both kidneys are smooth, dark red,

congested. Normal corticomedullary markings are noted on sagittal section. The calyces, pelves, ureters are normal. The urinary bladder contains 200 mL of clear yellow urine. The prostate and seminal vesicles are normal. The testes are normal to palpation. A normal circumcised penis is present.

GASTROINTESTINAL TRACT:
The esophageal mucosa is autolyzed. The gastric mucosa is autolyzed. The stomach contains 1130 grams of viscous, masticated, pale tan food material containing fragments of vegetable material and meat. The small and large bowel are grossly normal. The vermiform appendix is present and normal.

MUSCULOSKELETAL SYSTEM:
The musculoskeletal system normal. There is exogenous obesity and the abdominal panus is 4.5 cm in thickness.

URINE DRUG SCREEN:
Medtox Immunochromatographic plate

THC:	Negative.
Opiates:	Negative.
Amphetamines:	Negative.
Cocaine:	Negative.
PCP:	Negative.

CERTIFICATE OF DEATH &
AUTOPSY (AMENDED)

CERTIFICATE OF DEATH

STATE OF CALIFORNIA
USE BLACK INK ONLY / NO ERASURES, WHITEOUTS OR ALTERATIONS
VS-11(REV 1/04)

STATE FILE NUMBER | LOCAL REGISTRATION NUMBER

DECEDENT'S PERSONAL DATA

1. NAME OF DECEDENT — FIRST (Given)	2. MIDDLE	3. LAST (Family)
DANIEL	ELWIN	MCCORNACK

AKA. ALSO KNOWN AS — Include full AKA (FIRST, MIDDLE, LAST)	4. DATE OF BIRTH mm/dd/ccyy	5. AGE Yrs.	IF UNDER ONE YEAR Months	IF UNDER ONE YEAR Days	IF UNDER 24 HOURS Hours	IF UNDER 24 HOURS Minutes	6. SEX
- - -	02/15/1963	45					M

9. BIRTH STATE/FOREIGN COUNTRY	10. SOCIAL SECURITY NUMBER	11. EVER IN U.S. ARMED FORCES?	12. MARITAL STATUS (at Time of Death)	7. DATE OF DEATH mm/dd/ccyy	8. HOUR (24 Hours)
CA	555-51-7837	[ ] YES [X] NO [ ] UNK	MARRIED	03/23/2008	0052

13. EDUCATION — Highest Level/Degree (see worksheet on back)	14/15. WAS DECEDENT HISPANIC/LATINO(A)/SPANISH? (If yes, see worksheet on back.)	16. DECEDENT'S RACE — Up to 3 races may be listed (see worksheet on back)
HS GRADUATE	[ ] YES [X] NO	CAUCASIAN

17. USUAL OCCUPATION — Type of work for most of life. DO NOT USE RETIRED	18. KIND OF BUSINESS OR INDUSTRY (e.g., grocery store, road construction, employment agency, etc.)	19. YEARS IN OCCUPATION
PLANT MANAGER	CHEMICAL MANUFACTURE	25

USUAL RESIDENCE

20. DECEDENT'S RESIDENCE (Street and number or location): 6255 PEACHY CANYON RD.

21. CITY	22. COUNTY/PROVINCE	23. ZIP CODE	24. YEARS IN COUNTY	25. STATE/FOREIGN COUNTRY
PASO ROBLES	SAN LUIS OBISPO	93446	45	CA

INFORMANT

26. INFORMANT'S NAME, RELATIONSHIP	27. INFORMANT'S MAILING ADDRESS (Street and number or rural route number, city or town, state, ZIP)
KATHY MCCORNACK, WIFE	6255 PEACHY CANYON RD., PASO ROBLES, CA 93446

SPOUSE AND PARENT INFORMATION

28. NAME OF SURVIVING SPOUSE — FIRST	29. MIDDLE	30. LAST (Maiden Name)	
KATHY	MARIE	ESPARZA	
31. NAME OF FATHER — FIRST	32. MIDDLE	33. LAST	34. BIRTH STATE
RALPH	MICHAEL	MCCORNACK	CA
35. NAME OF MOTHER — FIRST	36. MIDDLE	37. LAST (Maiden)	38. BIRTH STATE
LINDA	EILEEN	HIRSCHLER	CA

FUNERAL DIRECTOR/ LOCAL REGISTRAR

39. DISPOSITION DATE mm/dd/ccyy	40. PLACE OF FINAL DISPOSITION	
03/28/2008	PASO ROBLES DISTRICT CEMETERY	45 NACIMIENTO LAKE DR., PASO ROBLES, CA 93446

41. TYPE OF DISPOSITION(S)	42. SIGNATURE OF EMBALMER	43. LICENSE NUMBER
BU	NOT EMBALMED	-

44. NAME OF FUNERAL ESTABLISHMENT	45. LICENSE NUMBER	46. SIGNATURE OF LOCAL REGISTRAR	47. DATE mm/dd/ccyy
KUEHL-NICOLAY FUNERALS AND CREM	FD68	POKI NAMKUNG, M.D.	03/27/2008

PLACE OF DEATH

101. PLACE OF DEATH	102. IF HOSPITAL, SPECIFY ONE	103. IF OTHER THAN HOSPITAL, SPECIFY ONE
CAMPSITE	[ ] IP [ ] ER/OP [ ] DOA	[ ] Hospice [ ] Nursing Home/LTC [ ] Decedent's Home [X] Other

104. COUNTY	105. FACILITY ADDRESS OR LOCATION WHERE FOUND (Street and number or location)	106. CITY
SANTA CRUZ	4770 SITE 1 HIGHWAY 9	FELTON

CAUSE OF DEATH

107. CAUSE OF DEATH — Enter the chain of events — diseases, injuries, or complications — that directly caused death. DO NOT enter terminal events such as cardiac arrest, respiratory arrest, or ventricular fibrillation without showing the etiology. DO NOT ABBREVIATE.

		Time Interval Between Onset and Death
IMMEDIATE CAUSE (Final disease or condition resulting in death) →	(A) CARDIAC ARREST	(A1) MINS
Sequentially, list conditions, if any, leading to cause on Line A. Enter UNDERLYING CAUSE (disease or injury that initiated the events resulting in death) LAST	(B) VENTRICULAR ARRHYTHMIA	(B1) MINS
	(C) ATRIAL FIBRILLATION	(C1) YEARS
	(D) HYPERTENSIVE AND ARTERIOSCLEROTIC CARDIOVASCULAR DISEASE	(D1) YEARS

108. DEATH REPORTED TO CORONER?	109. BIOPSY PERFORMED?	110. AUTOPSY PERFORMED?	111. USED IN DETERMINING CAUSE?
[X] YES [ ] NO	[ ] YES [X] NO	[X] YES [ ] NO	[X] YES [ ] NO
REFERRAL NUMBER 08-02790			

112. OTHER SIGNIFICANT CONDITIONS CONTRIBUTING TO DEATH BUT NOT RESULTING IN THE UNDERLYING CAUSE GIVEN IN 107: EXOGENOUS OBESITY

113. WAS OPERATION PERFORMED FOR ANY CONDITION IN ITEM 107 OR 112? (If yes, list type of operation and date.)	113A. IF FEMALE, PREGNANT IN LAST YEAR?
NO	[ ] YES [ ] NO [ ] UNK

PHYSICIAN'S CERTIFICATION

114. I CERTIFY THAT TO THE BEST OF MY KNOWLEDGE DEATH OCCURRED AT THE HOUR, DATE, AND PLACE STATED FROM THE CAUSES STATED.	115. SIGNATURE AND TITLE OF CERTIFIER	116. LICENSE NUMBER	117. DATE mm/dd/ccyy
Decedent Attended Since: (A) mm/dd/ccyy; Decedent Last Seen Alive: (B) mm/dd/ccyy	118. TYPE ATTENDING PHYSICIAN'S NAME, MAILING ADDRESS, ZIP CODE		

CORONER'S USE ONLY

119. I CERTIFY THAT IN MY OPINION DEATH OCCURRED AT THE HOUR, DATE, AND PLACE STATED FROM THE CAUSES STATED. MANNER OF DEATH	120. INJURED AT WORK?	121. INJURY DATE mm/dd/ccyy	122. HOUR (24 Hours)
[X] Natural [ ] Accident [ ] Homicide [ ] Suicide [ ] Pending Investigation [ ] Could not be determined	[ ] YES [ ] NO [ ] UNK		

123. PLACE OF INJURY (e.g., home, construction site, wooded area, etc.)

124. DESCRIBE HOW INJURY OCCURRED (Events which resulted in injury)

125. LOCATION OF INJURY (Street and number, or location, and city, and ZIP)

Exhibit 4
Wit: Mason
Date: 10/1/09
Allison Ash-Hoyman, CSR

126. SIGNATURE OF CORONER / DEPUTY CORONER	127. DATE mm/dd/ccyy	128. TYPE NAME, TITLE OF CORONER / DEPUTY CORONER
NAOMI SILVA	03/27/2008	NAOMI SILVA, DEPUTY CORONER

STATE REGISTRAR	A	B	C	D	E	Printed on: 09/30/2009 03:47 PM By BURT, ALAN (ABURT)	FAX AUTH. #	CENSUS TRACT

PHYSICIAN/CORONER'S AMENDMENT

DEATHS AFTER 1-1994

NO ERASURES, WHITEOUTS, OR OTHER ALTERATIONS
USE BLACK INK ONLY

STATE FILE NUMBER: 1.1

LOCAL REGISTRATION DISTRICT AND CERTIFICATE NUMBER:

PART I INFORMATION TO LOCATE RECORD

	1. NAME--FIRST (GIVEN)	2. MIDDLE	3. LAST (FAMILY)	4. SEX
NAME AS IT APPEARS ON RECORD	DANIEL	ELWIN	MCCORNACK	M
ADDITIONAL INFORMATION TO LOCATE RECORD	5. DATE OF EVENT--MM/DD/CCYY: 03/23/2008	6. CITY OF OCCURRENCE: FELTON	7. COUNTY OF OCCURRENCE: SANTA CRUZ	

PART II STATEMENT OF CORRECTIONS

LIST ONE ITEM PER LINE

8. CERTIFICATE ITEM NUMBER	9. INFORMATION AS IT APPEARS ON ORIGINAL RECORD	10. INFORMATION AS IT SHOULD APPEAR
107A	CARDIAC ARREST	CARDIAC ARRHYTHMIA
107B	VENTRICULAR ARRHYTHMIA	DIGOXIN TOXICITY
107BT	MINS	DAYS
107C	ATRIAL FIBRILLATION	DIGOXIN POISONING
107CT	YEARS	DAYS
107D	HYPERTENSIVE AND ARTERIOSCLEROTIC CARDIOVASCULAR DISEASE	
107DT	YEARS	
108A	08-02790	08-02797
112	EXOGENOUS OBESITY	HYPERTENSIVE AND ARTERIOSCLEROTIC CARDIOVASCULAR DISEASE AND EXOGENOUS OBESITY
119	NATURAL	ACCIDENT
120		NO
121		03/23/2008
122		0052
123		SMITH WOODS RV PARK
124		ACCIDENTAL OVERDOSE ON DIGOXIN
125		4770 HIGHWAY 9, FELTON, CA 95018

DECLARATION OF CERTIFYING PHYSICIAN OR CORONER

I HEREBY DECLARE UNDER PENALTY OF PERJURY THAT THE ABOVE INFORMATION IS TRUE AND CORRECT TO THE BEST OF MY KNOWLEDGE.

11. SIGNATURE OF CERTIFYING PHYSICIAN OR CORONER	12. DATE SIGNED--MM/DD/CCYY	13. TYPED OR PRINTED NAME AND TITLE/DEGREE OF CERTIFIER
▶ ALAN G BURT	09/30/2009	SUP DEPUTY CORONER

14. ADDRESS--STREET AND NUMBER	15. CITY	16. STATE	17. ZIP CODE
701 OCEAN STREET, RM 340	SANTA CRUZ	CA	95060

STATE/LOCAL REGISTRAR USE ONLY

18. OFFICE OF VITAL RECORDS OR SIGNATURE OF LOCAL REGISTRAR	19. DATE ACCEPTED FOR REGISTRATION--MM/DD/YY
▶	

STATE OF CALIFORNIA, DEPARTMENT OF HEALTH SERVICES, OFFICE OF VITAL RECORDS — VS 24A (REV. 10/03)

Printed on: 09/30/2009 03:46 PM
By BURT, ALAN (ABURT)

1.1

PLAINTIFFS' EXHIBITS 012548

5-198

SANTA CRUZ COUNTY SHERIFF-CORONER'S OFFICE
701 Ocean Street
Santa Cruz, California
* REPORT OF AUTOPSY EXAMINATION *

AUTOPSY NUMBER: CA08-037 **FILE NUMBER:** 08-02797

NAME: Daniel Mc Cornack **AGE:** 45 **SEX:** Male

PLACE OF DEATH: Smithwood R.V. Park, 4770 Hwy 9, Felton

DATE/HOUR OF DEATH: March 23, 2008 @ 0052 Hours

AUTOPSY PERFORMED: Santa Cruz County Morgue

DATE/HOUR OF AUTOPSY: March 26, 2008 @ 7:30 a.m.

PATHOLOGIST: Richard T. Mason, M.D.

BODY IDENTIFIED BY: Ankle tag.

ATTENDING PHYSICIAN: None.

CAUSE OF DEATH: CARDIAC ARREST
Due to: Ventricular arrhythmia
Due to: Digoxin toxicity
Due to: Digoxin poisoning.

CONTRIBUTORY: Exogenous obesity.

MANNER: ~~Natural.~~ Accident RM

DIAGNOSES:

1. Digoxin poisoning with:
   A. Toxic level of digoxin present in blood, 3.6nanog/mL.
   B. Cardiac arrhythmia due to digoxin toxicity.

Exhibit 5
Wit: Mason
Date: 10/1/09
Allison Ash-Hoyman, CSR

DIAGNOSES, continued

2. Hypertensive and arteriosclerotic cardiovascular disease with:
   A. Cardiomegaly and left ventricular hypertrophy.
   B. Coronary arteriosclerosis, mild to moderate.
   C. Myocardial fibrosis, mild.
   D. Atrial fibrillation by history.
   E. Probable ventricular arrhythmia and arrest.

3. Cerebral edema and congestion.

4. Pulmonary edema and congestion.

5. Exogenous obesity, moderate.

Richard T. Mason M.D.
RICHARD T. MASON, M.D.
Forensic Pathologist

RTM/dp

EXTERNAL EXAMINATION

The body examined is that of a well-developed, mildly obese, middle-aged white male that appears the stated age of 45 years. The body is 70 inches in length and weighs 220 pounds. The scalp hair is medium brown with gray and is cut short measuring 1/4 inch. The eyes are blue gray with the pupils equal in diameter, measuring 6 mm. There is an adhesive nostril dilating device attached over the midportion of the nostrils. There is a short 3/4 inch grayish brown mustache. Natural teeth in good condition are present in the mouth. There is a 1-2 mm growth of beard present on the lower face. There is prominent pinkish cyanosis of the anterior face and neck.

Examination of the anterior chest reveals 4 x 6 inch adhesive defibrillator electrodes present over the left lower lateral chest and the right upper anterior chest. Adhesive EKG electrodes are present over the right and left upper anterior chest and the right and left lower abdomen. The axillae are normal.

Examination of the anterior abdomen reveals it to be mildly obese. There is a slight umbilical hernia. There are no other marks or wounds are noted on the anterior abdomen. Normal male external genitalia are present. The penis is circumcised.

Examination of the lower limbs reveals normal, symmetric, muscular right and left thighs and right and left lower legs. There is a coroner's identification band present on the right ankle bearing the name: McCornack, Daniel; #08-2790. The right and left feet are normal.

Examination of the upper limbs reveals normal, symmetric, muscular right and left upper arms and right and left forearms.

The antecubital spaces are clean with no marks or wounds. The right and left forearms are unremarkable. An intravenous line is in position through a needle puncture wound on the dorsum of the left hand. This line is attached to a 1-liter bag of normal saline.

Examination of the hands reveals them to be normal with short intact fingernails.

INTERNAL EXAMINATION

HEAD:
Reflection of the scalp reveals an absence of any contusions on the galeal surface. The calvarium is intact. Reflection of the calvarium reveals prominent cerebral edema. The gyri are flattened. The meninges are clear but congested. The brain weighs 1,640 grams. The brain has a normal external morphology except for the edema. The cerebral arteries are normal in distribution and appearance.

Multiple coronal sections through both cerebral hemispheres reveal normal cortex, normal white matter and normal basal ganglia. Sections through the brainstem and cerebellum reveal these structures to be normal.

The dura is stripped from the base of the skull to reveal an intact skull base.

NECK:
The hyoid bone, larynx, trachea, soft tissues, cervical spine are intact. The airway is fully patent.

BODY CAVITIES:
The pericardial cavity contains 25 mL of clear yellow fluid. There is no excess fluid in the pleural or peritoneal cavities.

CARDIOVASCULAR SYSTEM:
Heart weight 500 grams. There is cardiomegaly and left ventricular hypertrophy. The epicardial surfaces are smooth and glistening. The heart valves are normal. The atria are normal in size. The endocardial surfaces of the atria and ventricles are normal in appearance. Dissection of the coronary arteries reveals abundant, scattered, flattened atherosclerotic plaque in the right coronary artery, which is of greatest circumference compared to the LAD and the circumflex coronary arteries. There is flattened atherosclerotic plaque in a small left anterior descending coronary artery. There is a minimal amount of atherosclerotic plaque in the left circumflex coronary artery. Multiple cross sections through both ventricles of the heart reveal some mild diffusely distributed myocardial fibrosis. There is cardiomegaly and left ventricular hypertrophy with the left ventricle measuring 16 mm in thickness and the right ventricle measuring 4 mm in thickness. There are no foci or evidence of old or recent myocardial infarction.

Examination of the aorta reveals it to be smooth with minimal focal atherosclerosis. The superior and inferior vena cavae are intact and normal with no thromboemboli.

RESPIRATORY TRACT:
Lungs, weight right 830 grams, left 840 grams. There is severe bilateral pulmonary edema and congestion. Bloodstained watery fluid runs from the cut surfaces of all lobes of both lungs. There are no foci of consolidation. The major bronchi contain a small amount of bloodstained edema fluid. The pulmonary arteries are widely patent with no thromboemboli.

LIVER:
Weight 2,550 grams. The smooth, light, reddish tan capsular surface is intact. The liver is enlarged and there is fatty metamorphosis. The parenchyma is light pinkish tan and fractures easily on digital pressure. There is no increase in fibrous tissues to palpation. The intra and extrahepatic blood vessels and bile ducts are grossly normal. The gallbladder is thin-walled and contains 1 mL of light brown transparent bile.

SPLEEN:
Weight 470 grams. This organ is enlarged and congested. The dark gray brown capsular surface is intact with no evidence of trauma. The parenchyma is dark red brown firm.

PANCREAS:
Weight 210 grams. Normal, pale tan, lobular, autolyzed parenchyma is noted on cut section.

ENDOCRINE SYSTEM:
The pituitary, adrenal and thyroid glands are grossly normal.

GENITOURINARY TRACT:
Kidneys, weight right 230 grams, left 220 grams. The cortical surfaces of both kidneys are smooth, dark red,

congested. Normal corticomedullary markings are noted on sagittal section. The calyces, pelves, ureters are normal. The urinary bladder contains 200 mL of clear yellow urine. The prostate and seminal vesicles are normal. The testes are normal to palpation. A normal circumcised penis is present.

GASTROINTESTINAL TRACT:
The esophageal mucosa is autolyzed. The gastric mucosa is autolyzed. The stomach contains 1130 grams of viscous, masticated, pale tan food material containing fragments of vegetable material and meat. The small and large bowel are grossly normal. The vermiform appendix is present and normal.

MUSCULOSKELETAL SYSTEM:
The musculoskeletal system normal. There is exogenous obesity and the abdominal panus is 4.5 cm in thickness.

URINE DRUG SCREEN:
Medtox Immunochromatographic plate

THC:	Negative.
Opiates:	Negative.
Amphetamines:	Negative.
Cocaine:	Negative.
PCP:	Negative.

NMS LABS

NMS LABS

NMS Labs
3701 Welsh Road, PO Box 433A, Willow Grove, PA 19090-0437
Phone: (215) 657-4900 Fax: (215) 657-2972
e-mail: nms@nmslabs.com
Robert A. Middleberg, PhD, DABFT, DABCC, Laboratory Director

CONFIDENTIAL

June 24, 2008

TO: 60C
Santa Cruz County Coroner
Attn: Alan Burt
701 Ocean Street, #340
Santa Cruz, CA 95060

SUPPLEMENTAL TOXICOLOGY REPORT OF: **McCORNACK, Daniel E.** **45/M**

NMS Workorder No: 08095896
NMS Control No: 10843208
Client ID No: 08-0279

SPECIMENS: One gray top tube containing ~ 10 mL of peripheral blood, one clear vial containing ~ 14 mL of peripheral blood and two white plastic containers (one containing ~ 30 mL of urine and one containing ~ 32 g of liver) were received on 03/28/08.

EXAMINATION: Analysis Requested - Panel 8092B - Autopsy Toxicology Therapeutic and Abused Drug Screen
Test No. 1615B – Digoxin

FINDINGS:

Blood

ETHYL ALCOHOL (by Enzymatic Assay & Headspace GC)	48 mg/dL (BAC=0.048 % w/v)
DILTIAZEM (by GC & GC/MS)	630 nanog/mL
DIGOXIN (by LC-MS/MS)	3.6 nanog/mL
QUINIDINE/QUININE* (by GC/MS)	Trace
ATROPINE (by GC/MS)	Positive

*Quinine and quinidine can be differentiated analytically, but this is a separate analysis. If further delineation is necessary, please contact the laboratory.

Incidental findings by GC/MS: CAFFEINE and THEOBROMINE.

Other than the above findings, examination of the specimens submitted did not reveal any positive findings of toxicological significance by procedures outlined in the accompanying Analysis Summary.

6.27.0

CONFIDENTIAL

NMS Workorder No: 08095896
NMS Control No: 10843208
Client ID No: 08-02790

COMMENTS:

1. Ethyl alcohol is a CNS-depressant and has effects so-related, e.g., impaired judgment, alertness and coordination.

   If the determined blood alcohol concentration (BAC) is representative of the circulating BAC at the time of the fatal incident, then it represents as absorbed body burden of approximately 2 "drinks" of an alcoholic beverage in an adult of average size weighing approximately 155 lbs.

   Note: a "drink" = 1 oz. of distilled spirits
   4 oz. of wine
   12 oz. of beer

   Each of the drinks listed above contains about the same amount of ethyl alcohol.

2. Diltiazem (Cardizem®) is a calcium channel blocking coronary vasodilator indicated for the treatment of variant, exertional and unstable angina. It is also used in arrhythmic and/or hypertensive therapy. Desacetyldiltiazem is an active metabolite of diltiazem. Divided doses up to 180-360 mg daily may be prescribed for angina.

   Therapeutic blood levels of diltiazem appear to be in the range of 50 to 200 nanog/mL. Numerous cases of diltiazem overdose have been reported. The majority of individuals who receive prompt treatment survive diltiazem overdose; however, death has been reported, especially in conjunction with other substances. Diltiazem has been found mixed with cocaine, either as a cutting agent or in an attempt to reduce cocaine-induced increased blood pressure. In a separate, small series of diltiazem related fatalities, the postmortem blood concentrations range from 6700 to 33,000 nanog/mL (mean 16,000 nanog/mL). In addition, diltiazem is reported to undergo postmortem redistribution with an average heart blood/femoral blood ratio of 2.6.

3. Digoxin (Lanoxin®) is a cardiac glycoside used in the treatment of congestive heart failure and other contractility-related deficiencies. There is considerable individualization of the dose of this medication and what is therapeutic in one individual may be toxic in another.

   Individuals are generally titrated to find an appropriate dosage, especially since digoxin has a low therapeutic index.

4. Quinine and quinidine are stereoisomers derived from the bark of the cinchona tree. Quinine has been used in the past as an antimalarial, but is more commonly used today to treat muscle cramps. It is also used as a flavoring agent in tonic waters and as a cutting agent adulterant in illicit street drug dosages of heroin. Adverse effects include gastrointestinal disturbances, tinnitus, dizziness, arrhythmias and hypotension.

   Quinidine is frequently used as an antiarrhythmic agent. It is available for acute administration by intramuscular or intravenous injection of 200 to 750 mcg or for maintenance therapy in oral doses of 600 to 4,000 mg daily. . Toxicity is manifested by gastrointestinal disturbances, giddiness, tinnitus, diplopia and hypotension.

5. Atropine is an anticholinergic alkaloid used in pre-anesthetic therapy to control airway secretions and as an antispasmodic to control gastrointestinal spasms. It is frequently used as an antidote in the treatment of anticholinesterase-type pesticides. It can be obtained naturally from deadly nightshade or jimson weed. Atropine is also used in resuscitative attempts.

   Toxic effects of atropine have considerable individual variation; however, at high doses, signs and symptoms include mydriasis, hot dry reddened skin, deliriums and hallucinations.

   In resuscitative failure, most of the administered drug remains confined to the intravascular injection pathway.

NMS
LABS

NMS Labs
3701 Welsh Road, PO Box 433A, Willow Grove, PA 19090-0437
Phone: (215) 657-4900 Fax: (215) 657-2972
e-mail: nms@nmslabs.com
Robert A. Middleberg, PhD, DABFT, DABCC, Laboratory Director

CONFIDENTIAL

May 29, 2009

RECEIVED
JUN 04 2009
ERNST & MATTISON

TO: M60112
Ernst & Mattison
1020 Palm Street
San Luis Obispo, CA 93401

CRIMINALISTICS REPORT OF: **McCORNACK SR., Daniel Elwin**
NMS Workorder No: 09107925
Client ID No: Not Provided

SPECIMENS: Item 1 One clear plastic container containing one white pill monogrammed "B-146"

The above evidence was received from United Parcel Services on 05/14/09.

EXAMINATION: Analysis Requested – Test No. 7011 – Special Request for Digoxin

FINDINGS:

Item 1

DIGOXIN (by LC-MS/MS)	0.250 mg/tablet
WEIGHT	113.79 mg
THICKNESS	Not measured due to pill being broken

Respectfully,

Matthew McMullin, MS, DABFT
Forensic Toxicologist

MMM/sdw

This analysis was performed under chain of custody. The chain of custody documentation is on file at NMS Labs.

The remainder of the submitted specimens are scheduled to be returned/discarded six (6) weeks from the date of this report unless alternate arrangements are made by you prior thereto.

PLT DEM,Sr.(2)00082

PLAINTIFFS' EXHIBITS 012559

5-209

CONFIDENTIAL

NMS Workorder No: 08095896
NMS Control No: 10843208
Client ID No: 08-02790

Respectfully,

Edward J. Barbieri, Ph.D.
Forensic Toxicologist

EJB/ltb

This analysis was performed under chain of custody. The chain of custody documentation is on file at NMS Labs.

Unless alternate arrangements are made by you, the remainder of the submitted specimens will be discarded six (6) weeks from the date of this report; and generated data will be discarded five (5) years from the date of this report.

****** **** ANALYSIS SUMMARY **** ******

8092B - Therapeutic and Abused Drug Screen

Test No. 8092B – Drug Screen by Enzyme-Linked Immunosorbent Assay (ELISA) on Blood for: Amphetamine, Barbiturates, Benzodiazepines, Cannabinoids (Marihuana), Cocaine/Metabolites, Methamphetamine, Opiates and Phencyclidine (PCP); Headspace Gas Chromatography for Ethanol, Methanol, Acetone and Isopropyl Alcohol.

Test No. 8092B - Drug Screen II- Gas Chromatography and Gas Chromatography/Mass Spectrometry Analysis on Blood:

The following is a general list of compound classes included in the Gas Chromatographic screen. Other specific compounds outside these classes are also included. Please note that not all known compounds included in each specified class or heading are included. The detection of any particular compound is concentration-dependent. For a detailed list of all compounds included in this screen, please contact NMS Labs.

Analgesics (opioid and non-opioid), Anesthetics, Antiasthmatic Agents, Anticholinergic Agents, Anticonvulsant Agents, Antidepressants, Antiemetic Agents, Antihistamines, Antiparkinsonian Agents, Antipsychotic Agents, Antitussive Agents, Anxiolytics (Benzodiazepine and others), Cardiovascular Agents (non-digitalis), Hallucinogens, Hypnosedatives (Barbiturate and others), Muscle Relaxants, Non-Steroidal Anti-Inflammatory Agents (excluding Salicylate) and Stimulants (Amphetamine-like and others).

Test No. 8092B - Colorimetric Analysis on Blood for: Salicylates and Acetaminophen.

Test No. 5010B - Alcohol Confirmation - Enzymatic Assay on Blood for: Ethanol (Ethyl alcohol).

Test No. 1640B - Diltiazem - Gas Chromatography on Blood for: Diltiazem.

Test No. 1615B – Digoxin – Liquid Chromatography - Tandem Mass Spectrometry on Blood for: Digoxin.

******* END OF REPORT *******

CONFIDENTIAL

NMS Workorder No: 09107925
Client ID No: Not Provided

***** *****ANALYSIS SUMMARY***** *****

Test No. 7011 – Special Request - Liquid Chromatography – Tandem Mass Spectrometry on Pills for: Digoxin.

***** END OF REPORT *****

NMS LABS

NMS Labs
3701 Welsh Road, PO Box 433A, Willow Grove, PA 19090-0437
Phone: (215) 657-4900 Fax: (215) 657-2972
e-mail: nms@nmslabs.com
Robert A. Middleberg, PhD, DABFT, DABCC, Laboratory Director

CONFIDENTIAL

September 22, 2009

RECEIVED
SEP 25 2009
ERNST & MATTISON

TO: M60112
Ernst & Mattison
Attn: Terry Kilpatrick
1020 Palm Street
San Luis Obispo, CA 93401

SUPPLEMENTAL CRIMINALISTICS REPORT OF: McCORNACK SR., DANIEL ELWIN
NMS Workorder No: 09154008
Client ID No: Prior NMS Workorder No: 09107925

SPECIMENS: Item 1 Five white pills in a Cinnamon Altoids® container.

The above evidence was received from United State Postal Service Priority Mail on 07/13/09.

EXAMINATION: Analysis Requested – Test No. 7011 – Special Request for Digoxin

FINDINGS:

Item 1.a

DIGOXIN (by LC-MS/MS)	0.247 mg/pill
WEIGHT	122.939 mg
THICKNESS	3.28 mm

Item 1.b

DIGOXIN (by LC-MS/MS)	0.244 mg/pill
WEIGHT	127.597 mg
THICKNESS	3.56 mm

Item 1.c

DIGOXIN (by LC-MS/MS)	0.227 mg/pill
WEIGHT	129.432 mg
THICKNESS	3.59 mm

CONFIDENTIAL

NMS Workorder No: 09154008
Client ID No: Prior NMS Workorder No: 09107925

Item 1.d

DIGOXIN (by LC-MS/MS)	0.227 mg/pill
WEIGHT	128.525 mg
THICKNESS	3.54 mm

Item 1.e

DIGOXIN (by LC-MS/MS)	0.261 mg/pill
WEIGHT	127.690 mg
THICKNESS	3.52 mm

Respectfully,

Matthew McMullin, MS, DABFT
Forensic Toxicologist

MMM/sdw

This analysis was performed under chain of custody. The chain of custody documentation is on file at NMS Labs.

The remainder of the submitted specimens are scheduled to be returned/discarded six (6) weeks from the date of this report unless alternate arrangements are made by you prior thereto.

***** ***** **ANALYSIS SUMMARY** ***** *****

Test No. 7011 – Special Request – Liquid Chromatography – Tandem Mass Spectrometry on Pill for: Digoxin.

***** **END OF REPORT** *****

CVS Caremark
Recall Letter

CVS CAREMARK | CAREMARK®

May 2008

Dear Plan Participant:

You recently received a letter from CVS Caremark about the Digitek® (digoxin tablets, USP) 0.125 mg and Digitek (digoxin tablets, USP) 0.25 mg Patient Level Recall. We are providing you with an important update.

Please be aware that as a result of this recall, there is a market-wide shortage of digoxin. In an effort to meet the needs of all plan participants, enclosed is a maximum of a 45-day supply of replacement product.

What to Do with Your Digitek
For your safety and to ensure proper disposal, we have provided you with a return envelope. Please send your Digitek tablets to CVS Caremark in the original prescription bottle, if possible.

We encourage you to contact your provider with any questions or concerns regarding continuation of therapy.

Sincerely,

CVS Caremark

Enclosure

For more information on this issue you may contact the U.S. Food and Drug Administration (FDA) consumer inquiry line toll-free at 1-888-INFO-FDA (1-888-463-6332) or by accessing the FDA Web site at www.fda.gov.

5-215

Recall -- Firm Press Release

FDA posts press releases and other notices of recalls and market withdrawals from the firms involved as a service to consumers, the media, and other interested parties. FDA does not endorse either the product or the company.

Actavis Totowa (formerly known as Amide Pharmaceutical, Inc.) recalls all lots of Bertek and UDL Laboratories Digitek® (digoxin tablets, USP) as precaution

Contact:
Stericycle customer service
1-888-276-6166

FOR IMMEDIATE RELEASE -- Morristown, NJ -- April 25, 2008 -- Actavis Totowa LLC, a United States manufacturing division of the international generic pharmaceutical company Actavis Group, is initiating a Class I nationwide recall of Digitek® (digoxin tablets, USP, all strengths) for oral use. The products are distributed by Mylan Pharmaceuticals Inc., under a "Bertek" label and by UDL Laboratories, Inc. under a "UDL" label.

The voluntary all lot recall is due to the possibility that tablets with double the appropriate thickness may have been commercially released. These tablets may contain twice the approved level of active ingredient than it appropriate.

Digitek® is used to treat heart failure and abnormal heart rhythms. The existence of double strength tablets poses a risk of digitalis toxicity in patients with renal failure. Digitalis toxicity can cause nausea, vomiting, dizziness, low blood pressure, cardiac instability and bradycardia. Death can also result from excessive Digitalis intake. Several reports of illnesses and injuries have been received.

Actavis manufactures the products for Mylan and the products are distributed by Mylan and UDL under the Bertek and UDL labels. Bertek and UDL are affiliates of Mylan.

Any customer inquiries related to this action should be addressed to Stericycle customer service at 1-888-276-6166 with representatives available Monday through Friday, 8 am to 5 pm EST. Additional information about the voluntary recall can also be found at www.actavis.us.

Retailers who have this product are urged to return the product to their place of purchase. If consumers have medical questions, they should contact their health care providers.

This recall is being conducted with the knowledge of the Food and Drug Administration.

Any adverse reactions experienced with the use of this product, and/or quality problems should also be reported to the FDA's MedWatch Program by phone at 1-800-FDA-1088, by Fax at 1-800-FDA-0178, by mail at MedWatch, FDA, 5600 Fishers Lane, Rockville, MD 20852-9787, or on the MedWatch website at www.fda.gov/medwatch.

#

PRESS RELEASES

09.05.2008 / Product

Digitek® recall – customer support and frequently asked questions

On April 25, Actavis Totowa LLC initiated a Class 1 nationwide recall of Digitek® (digoxin tablets, USP, all strengths) for oral use. The products, manufactured by Actavis, are distributed by Mylan Pharmaceuticals, Inc. under the "Bertek" label and by UDL Laboratories, Inc. under the "UDL" label.

Press release from April 25

Acting together with Actavis to enact the recall of the product, Mylan Pharmaceuticals, Inc. as the distributor of Digitek, retained Stericycle customer service center (tel. 1-888-276-6166) to act as the recall coordinator immediately following Actavis' decision to recall the product.

Please direct all inquiries you may have regarding this recall to Stericycle for proper handling and distribution.

Representatives at the service center are available to support all consumer queries and to provide direction on how to return your product.

If you have any questions about your treatment, or any medical inquiries regarding the product or possible substitutes, you should contact your physician immediately.

Frequently asked questions:

- **Q: Why is Actavis recalling Digitek® (digoxin)?**
  A: This voluntary all-lot recall is due to the possibility that some tablets with double the appropriate thickness may have been commercially released. These tablets may contain twice the approved level of active ingredient than is appropriate.
- **Q: What should I do if I'm taking Digitek® (digoxin)?**
  A: Patients should contact their physician to get a new prescription. All inquiries related to returning your product or the recall should be addressed to Stericycle customer service at **1-888-276-6166** (option 1 for consumers or option 2 for wholesalers/distributors and retailers.
  Representatives are available **Monday through Friday, 8 am to 5 pm EST.**
- **Q: How can I report serious side effects from, or reactions to, digoxin?**
  A: If you wish to report an adverse experience from taking digoxin, you are directed to select option "3" from the menu provided on the Stericycle answering service where you will be directed to an Actavis representative who will process your information. We also report such incidences to the FDA in accordance with Federal Regulations. Please call customer service at 1-888-276-6166, extension 3.
  Representatives are available Monday through Friday, 8 am to 5 pm EST.
  If you need medical attention or have any inquiries regarding your treatment, however, you should contact your physician.
- **Q: What is Digitek® (digoxin)?**
  A: Digitek is the brand name for digoxin tablets, USP 0.125 mg and 0.25 mg, manufactured by Actavis and distributed by Mylan Pharmaceuticals, Inc. under the Bertek and UDL labels. It is prescribed for oral use to treat heart failure and abnormal heart rhythms.
- **Q: Where is Digitek® (digoxin) manufactured?**
  A: Digitek is manufactured in the United States by Actavis Totowa, in New Jersey.
- **Q: Is the recall just in the United States, or also in other countries?**
  A: The recall is only in the United States.

Drugs

Facts and Myths about Generic Drugs

Today, 7 in 10 prescriptions filled in the United States are for generic drugs. This fact sheet explains how generic drugs are made and approved and debunks some common myths about these products.

FACT: FDA requires generic drugs to have the same quality and performance as the brand name drugs.

- When a generic drug product is approved, it has met rigorous standards established by the FDA with respect to identity, strength, quality, purity and potency. Some variability can and does occur during manufacturing, for both brand name and generic drugs. When a drug, generic or brand name, is mass produced, very small variations in purity, size, strength and other parameters are permitted. FDA puts limits on how much variability in composition or performance of a drug is acceptable.
- Generic drugs are required to have the same active ingredient, strength, dosage form, and route of administration as the brand name (or reference) product. Generic drugs do not need to contain the same inactive ingredients as the brand product.
- Through review of bioequivalence data, FDA assures that the generic product will perform the same as its respective brand name (or reference) product. This standard applies to all generic drugs, whether immediate or controlled release.
- A generic drug must be shown to be bioequivalent to the reference drug; that is, it must be shown to give blood levels that are very similar to those of the reference product. If blood levels are the same, the therapeutic effect will be the same. In that case, there is no need to carry out a clinical effectiveness study and they are not required.
- All generic manufacturing, packaging and testing sites must pass the same quality standards as those of brand name drugs and the generic products must meet the same exacting specifications as any innovator brand name product. In fact, many generic drugs are made in the same plants as innovator brand name drug products.
- If an innovator of a brand name drug switches drug production to an alternative manufacturing site, or they change formulation of their brand name drug, these companies are held to the same rigorous manufacturing requirements as those that apply to generic drug companies.

FACT: Research shows that generics work just as well as brand name drugs.

- A recent study evaluated the results of 38 published clinical trials that compared cardiovascular generic drugs to their brand-name counterparts. There was no evidence that brand-name heart drugs worked any better than generic heart drugs. [Kesselheim et al. Clinical equivalence of generic and brand-name drugs used in cardiovascular disease: a systematic review and meta-analysis. JAMA. 2008;300(21)2514-2526].

FACT: When it comes to price, there is a big difference between generic and brand name drugs. On average, the cost of a generic drug is 80 to 85% lower than the brand name product.

- An IMS National Prescription Audit shows that a typical formulary now charges $6 for generic medications, $29 for preferred branded drugs, and $40 or more for non-preferred branded drugs. [Aitken et al. Prescription drug spending trends in the United States: looking beyond the turning point. Health Aff (Millwood). 2009;28(1):w151-60].
- Independent research has shown that total prescription drug expenditures in the United

5-219

States only increased by 4.0% from 2006 to 2007, with total spending rising from $276 billion to $287 billion. This is a sharp decrease from the 8.9% growth rate observed in prescription drug expenditures in 2006. One factor cited as a reason for the slowdown is an increase in availability and use of generic drugs [Hoffman et al. Projecting future drug expenditures--2009. Am J Health Syst Pharm. 2009;66(3):237-57].

Recently, some misinformation has raised concerns over generic drugs. Below are some common myths in circulation.

MYTH: FDA lets generic drugs differ from the brand name counterpart by up to 45 percent.

FACT: This claim is false. Anyone who repeats this myth does not understand how FDA reviews and approves generic drugs.

- FDA recently evaluated 2,070 human studies conducted between 1996 and 2007. These studies compared the absorption of brand name and generic drugs into a person's body. These studies were submitted to FDA to support approval of generics. The average difference in absorption into the body between the generic and the brand name was **only 3.5 percent** [Davit et al. Comparing generic and innovator drugs: a review of 12 years of bioequivalence data from the United States Food and Drug Administration. Ann Pharmacother. 2009;43 (10):1583-97]. Some generics were absorbed slightly more, some slightly less. This amount of difference would be expected and acceptable, whether for one batch of brand name drug tested against another batch of the same brand, or for a generic tested against a brand name. In fact, there have been studies in which branded drugs were compared with themselves as well as with a generic. As a rule, the difference for the generic-to-brand comparison was about the same as the brand-to-brand comparison.
- Any generic drug modeled after a single, brand name drug (the reference) must perform approximately the same in the body as the brand name drug. There will always be a slight, but not medically important, level of natural variability – just as there is for one batch of brand name drug to the next.

MYTH: People who are switched to a generic drug are risking treatment failure.

FACT: There is no evidence for this claim. Treatment failures can and do occur when taking generic or brand name drugs. If someone is switched to a generic drug around the time they are relapsing, they may attribute the problem to the switch.

- Many people who have recovered from major depression have a relapse despite continued treatment. These relapses have been shown in trials of long-term therapy. [Byrne and Rothschild. Loss of antidepressant efficacy during maintenance therapy: possible mechanisms and treatments. J Clin Psychiatry. 1998;59(6):279-88].
- Many people who are on a seizure medications will re-experience a seizure despite continued treatment. [Randomised study of antiepileptic drug withdrawal in patients in remission. Medical Research Council Antiepileptic Drug Withdrawal Study Group. Lancet. 1991;337 (8751):1175-80].

- A percentage of people will re-experience gastric ulcers, despite an initial, positive response to and continued treatment with prescription strength antacids (cimetidine tablets; http://dailymed.nlm.nih.gov/dailymed/drugInfo.cfm?id=8131#nlm34067-9).

MYTH: Generic drugs cost less because they are inferior to brand name drugs.

FACT: Generic manufacturers are able to sell their products for lower prices, not because the products are of lesser quality, but because generic manufacturers generally do not engage in costly advertising, marketing and promotion, or significant research and

5-220

development.

- When a brand name drug comes off patent and generic drugs are permitted to compete with the brand name drug, the generic products compete by offering lower prices. Unlike the manufacturers of brand name drugs, generic drug companies do not have significant expenses to recoup for advertising, marketing and promotion, or research and development activities.

MYTH: There are quality problems with generic drug manufacturing. A recent recall of generic digoxin (called Digitek) shows that generic drugs put patients at risk.

FACT: FDA's aggressive action in this case demonstrates the high standards to which all prescription drugs – generic and brand name – are held.

- In March 2008, FDA performed a scheduled inspection of the Actavis production facility and identified products that were not manufactured to required specifications over a period of time extending back to the year 2006. Included in this list of products was one particular lot of Digitek.
- Actavis detected a very small number of oversized tablets in this lot (specifically, 20 double-sized tablets in a sample of approximately 4.8 million tablets).
- Although Actavis attempted to remove the affected Digitek tablets through visual inspection, FDA determined that this method of removal was inadequate to assure the product's quality and consistency in accordance with the current Good Manufacturing Practice (cGMP) regulations.
- Since the detection of the manufacturing problem, FDA has been actively engaged with this company to ensure that **ALL** potentially affected lots of Digitek tablets have been recalled. In our best judgment, given the very small number of defective tablets that may have reached the market and the lack of reported adverse events before the recall, harm to patients was very unlikely.
- FDA takes action whenever we find that a drug manufacturer is not following cGMPs. Over the last ten years, FDA has taken enforcement action against many brand name and generic firms for failing to meet FDA manufacturing quality standards.

MYTH: FDA's enforcement action against the generic drug company Ranbaxy demonstrates quality problems with imported generic drugs.

FACT: FDA's action demonstrates FDA's commitment to safe generic drugs.

- FDA has taken several regulatory actions against the generic drug manufacturer Ranbaxy, on the basis of problems at two of Ranbaxy's manufacturing facilities. Ranbaxy is one of many non-U.S. based generic and brand drug manufacturers.
- On Sept. 2008, the FDA issued two warning letters and instituted an Import Alert barring the entry of all finished drug products and active pharmaceutical ingredients from Ranbaxy's Dewas, Paonta Sahib and Batamandi Unit facilities due to violations of U.S. cGMP requirements. That action barred the commercial importation of 30 different generic drugs into the United States and remains in effect today (http://www.fda.gov/NewsEvents/Newsroom/PressAnnouncements/ucm149532.htm).
- Subsequent FDA investigations also revealed a pattern of questionable data raising significant questions regarding the reliability of certain generic drug applications from Ranbaxy.
- To address the allegedly falsified data, the FDA has invoked its Application Integrity Policy (AIP) against the Paonta Sahib facility. When the AIP is implemented, the FDA stops all substantive scientific review of any new or pending drug approval applications that contain data generated by the Paonta Sahib facility. This AIP covers applications that rely on data generated by the Paonta Sahib facility only.
- In the fiscal year 2008, FDA performed 2,221 drug-related inspections. FDA takes many

5-221

different enforcement actions, not just against generic drug manufacturers. For a list of enforcement actions in the fiscal year 2008, see http://www.fda.gov/downloads/ICECI/EnforcementActions/EnforcementStory/UCM129812.pdf. It is FDA's responsibility to ensure that the drugs people use, generic or brand name, are safe and effective.

MYTH: Brand name drugs are safer than generic drugs.

FACT: FDA receives very few reports of adverse events about specific generic drugs. Most reports of adverse events are related to side effects of the drug ingredient itself.

- The monitoring of postmarket adverse events for all drug products, including generic drugs, is one aspect of the overall FDA effort to evaluate the safety of drugs after approval. In most cases, reports of adverse events generally describe a **known reaction** to the active drug ingredient.

MYTH: FDA does not care about concerns over generic drugs.

FACT: FDA is actively engaged in making all regulated products – including generic drugs – safer.

- We are aware that there are reports noting that some people may experience an undesired effect when switching from brand name drug to a generic formulation or from one generic drug to another generic drug. Evidence indicates that if problems with interchangeability of drug formulations occur, they occur only for a very small subset of people.
- FDA is encouraging the generic industry to investigate whether, and under what circumstances, such problems occur. The Agency does not have the resources to perform independent clinical studies, and lacks the regulatory authority to require industry to conduct such studies. FDA will continue to investigate these reports to ensure that it has all the facts about these treatment failures and will make recommendations to healthcare professionals and the public if the need arises.

5-222

TWIN CITIES
COMMUNITY HOSPITAL
1100 LAS TABLAS ROAD
TEMPLETON, CA 93465

CLINICAL LABORATORY
C.L. DOUGLAS M.D., DIRECTOR
JAMES B. HANNA M.D. STEVEN B. JOBST M.D.
DAVID M. LAWRENCE M.D.
LAB PHONE 805 / 434-4501

Patient Name MCCORNACK, DANIEL E Birthdate 02/15/63 Sex M Location LA
MED RECORD # (0000)0103430
Admit Phys LEMM, GORDON MD Consulting Phys VONDOLLEN, L. MD INT

LAST DOSE 03-24-95 0730

DATE/TIME COLLECTED	PROCEDURE	UNITS	LOW	NORMAL	HIGH	REFERENCE RANGE
03/24/95 1630						
	T4	UG/DL		8.9		(4.2-11.8)
	T3 UPTAKE	%		34.5		(27.8-40.7)
	T7			3.1		(1.4-4.6)
	TSH	mIU/ML		1.56		(.32-5.00)
03/24/95 1630						
	DIGOXIN	NG/ML		1.4 f		(0.8-2.0)

DIGOXIN..... ——— —— ADULTS: < 0.5 NG/ML LIKELY INDICATES UNDERDIGITALIZATION.
THERAPEUTIC: 1.0-2.0 NG/ML.
TOXIC: MORE THAN 3.0 NG/ML.

EXHIBIT
6
Heard 7/14/11
PENGAD 800-631-6989

Footnotes
f = Footnote

Patient Name MCCORNACK, DANIEL E Printed 03/27/95 08:14 Page 1 END OF REPORT

2477 (7/94)

DEMGL:0090

6

Printed 08/02/01 Time 0632 TWIN CITIES COMMUNITY HOSPITAL
1 Clinical Laboratory
1100 Las Tablas Road
Templeton, California 93465
C.L.Douglas M.D ..Director

OUTPATIENT FINAL REPORT

Patient Name MCCORNACK, DANIEL E Medical Record Number (0000)0103430 Birthdate 02/15/1963
Nursing Station LA Room Number
Admit Phys LEMM, GORDON MD Consulting WATSON,D(959LASTABL) Referring YAMAGATA, NELSON
MISSION MEDICAL CLIN HIST #

LAST DOSE 7-31 0500

CHEMISTRY

Procedure	Ref Range	Unit	Specimen Date 08/01/01 Specimen Time 0855 Weekday/Day of Stay WED 002
URIC ACID SERUM	(3.4-7.0)	MG/DL	10.2 H

THERAPEUTIC DRUGS/TOXICOLOGY/ANTIBIOTIC LEVELS

Procedure	Ref Range	Unit	Specimen Date 08/01/01 Specimen Time 0855 Weekday/Day of Stay WED 002
DIGOXIN	(0.8-2.0)	NG/ML	1.7

GLYCEMIA STUDIES

08/01/01 0855
GLYCOHEMOGLOBIN 4.8 f %
GLYCOHEMOGLOBIN (07/19/01 -- Current)

Expected Range: 4.4% to 5.8%

Values less than 7.0% meet the treatment goal of the American Diabetes Association (ADA) for patients with Diabetes Mellitus. The ADA suggests additional action for values greater than 8.0%.

...notes
= High, f = Footnote

WIN CITIES COMMUNITY HOSPITAL
Clinical Laboratory
1100 Las Tablas Road
Templeton, California 93465
C.L.Douglas M.D.,Director
James B. Hannah, M.D. Steven B. Jobst, M.D. David M. Lawrence, M.D.
PATHOLOGISTS

MCCORNACK, DANIEL E
Birthdate 02/15/1963
Nursing Station LA Room Number
Admitting LEMM, GORDON MD
Referring LEMM, GORDON MD

Medical Record (0000)0103430
Account Number 0019810
Consulting VONDOLLEN, L. MD

CHEMISTRY

			Specimen Date	11/14/02
			Specimen Time	0840
			Weekday/Day of Stay	THU 001
Procedure	Ref Range	Unit		
ROUTINE CHEMISTRY				
GLUCOSE RANDOM	(70-110)	MG/DL	130	H
UREA NITROGEN	(8-21)	MG/DL	24	H
CREATININE	(.9-1.5)	MG/DL	1.4	
SODIUM	(134-145)	MEQ/L	141	
POTASSIUM	(3.5-5.1)	MEQ/L	4.2	
CHLORIDE	(98-107)	MEQ/L	100	
TOTAL CO2	(21.0-31.0)	MEQ/L	31.1	H
ANION GAP			14.1	
URIC ACID	(3.4-7.0)	MG/DL	8.2	H
CALCIUM	(8.4-10.4)	MG/DL	10.5	H
TOTAL PROTEIN	(6.0-8.3)	GM/DL	7.6	
ALBUMIN	(3.5-5.0)	GM/DL	4.6	
ALK PHOS	(45-122)	IU/L	76	
SGOT(AST)	(10-34)	IU/L	33	
SGPT(ALT)	(10-44)	IU/L	82	H
BILIRUBIN TOTAL	(0.2-1.3)	MG/DL	1.0	

THERAPEUTIC DRUGS/ TOXICOLOGY

			Specimen Date	11/14/02
			Specimen Time	0840
			Weekday/Day of Stay	THU 001
Procedure	Ref Range	Unit		
DIGOXIN	(0.8-2.0)	NG/ML	1.5	

MCCORNACK, DANIEL E 11/15/02 0344 Page 1 END OF REPORT

[FAXED REPORTS ARE CONFIDENTIAL AND INTENDED FOR PHYSICIAN ONLY.
IF RECEIVED IN ERROR. PLEASE CALL (805) 434-4501]

Patient Name MCCORNACK, DANIEL E Medical Record Number (0000)0103430
Nursing Station LA Room
Admitting LEMM - Do NOT Fax--> Referring VONDOLLEN, L. MD

CARDIAC RISK PROFILE

Procedure	Ref Range	Unit	Specimen Date 02/20/04 Specimen Time 0820 Weekday/Day of Stay FRI 002
CHOL/HDL RATIO			5.8 Hf

CHOL/HDL RATIO (07/23/01 -- Current)

CORONARY HEART DISEASE RISK	CHOLESTEROL/HDL RATIO
1/2 STANDARD RISK	3.4
STANDARD RISK FEMALE	4.4
STANDARD RISK MALE	5.0
2X STANDARD RISK FEMALE	7.1
2X STANDARD RISK MALE	9.6
3X STANDARD RISK FEMALE	11.0
3X STANDARD RISK MALE	23.4

LOW DENSITY LIPID (LDL) CALCULATION IS INVALID FOR SPECIMENS WITH A TOTAL TRIGLYCERIDE VALUE OF >400MG/DL

*LDL VALUES 130-159 (BORDERLINE RISK)
* LDL VALUES >160 (HIGH RISK)

THERAPEUTIC DRUGS/ TOXICOLOGY

Procedure	Ref Range	Unit	Specimen Date 02/20/04 Specimen Time 0820 Weekday/Day of Stay FRI 002
DIGOXIN	(0.8-2.0)	NG/ML	1.8

SPECIAL CHEMISTRY

Procedure	Ref Range	Unit	Specimen Date 02/20/04 Specimen Time 0820 Weekday/Day of Stay FRI 002
T4	(4.0-12.0)	UG/DL	7.5
TSH	(.32-5.00)	mIU/ML	3.24

CENTRAL COAST CLINICAL LAB
350 POSADA LANE STE 100
TEMPLETON, CA 93465
805 434-9080 FAX: 805 434-9082
DIRECTOR: CARL E. JOHNSON, JR. M.D.

NAME: MCCORNACK, DAN
PT ID:
PHYS1: LEMM, GORDON
292 POSADA LN STE D
PHYS2:

SEX: M
LAB ID: 021563DM
DRAW DATE: 07/28/06 08:10
PRINTED: 07/28/06 16:13
ACCESSION: 06209013

COMMENTS: FASTING
TESTS ORDERED: NOTE, HEMO, CMP, URCA, DIG, PSA, LIPID

PROCEDURE	IN RANGE	OUT OF RANGE		REFERENCE RANGE	
NOTE:	* LAST DOSE TAKEN: 7/27, 2200				
HEMOGRAM & PLT, AUTO					
WHITE CELL COUNT		12.6	H	3.8-10.6	10^3/cmm
RED CELL COUNT	5.79			4.70-5.90	10^6/cmm
HEMOGLOBIN	17.7			13.0-18.0	g/dL
HEMATOCRIT		52.4	H	42.0-52.0	%
MCV	91			80-100	um^3
MCH	30.6			24.0-34.0	Pg
MCHC	33.8			31.0-37.0	g/dL
RDW	12.3			11.5-14.0	%
PLATELET CNT	158			150-400	10^3/cmm
COMP. METABOLIC PANEL					
SODIUM	140			136-145	mEq/L
POTASSIUM	4.3			3.5-5.1	mEq/L
CHLORIDE	101			97-107	mEq/L
CARBON DIOXIDE	21			21-31	mEq/L
ANION GAP		22	H	10-20	
ALBUMIN	4.9			4.2-5.3	g/dL
PROTEIN, SERUM	6.9			6.0-8.3	g/dL
CALCIUM	10.0			8.4-10.5	mg/dL
BILIRUBIN, TOTAL	0.7			0.1-1.2	mg/dL
UREA NITROGEN, BLOOD		25	H	10-21	mg/dL
CREATININE, SERUM	1.1			0.6-1.3	mg/dL
ALK. PHOSPHATASE	62			41-111	U/L
ALT (SGPT)	46			0-46	U/L
AST (SGOT)	19			9-42	U/L
GLUCOSE	88			70-105	mg/dL
URIC ACID, SERUM		7.6	H	3.5-7.2	mg/dL
DIGOXIN	1.5			0.5-2.0	ng/mL
PSA, TOTAL	0.55			(4.0	ng/mL

END OF PAGE 1. CONTINUED ON PAGE 2

Needs fm -E

CENTRAL COAST CLINICAL LAB
350 POSADA LANE STE 100
TEMPLETON, CA 93465
805 434-9080 FAX: 805 434-9082
DIRECTOR: CARL E. JOHNSON, JR. M.D.

NAME: MCCORNACK, DAN
PT ID:
PHYS1: LEMM, GORDON
292 POSADA LN STE D
PHYS2: LAWRENCE VONDOLLEN
FAX 782-8859

SEX: M DOB: 02/15/1963
LAB ID: 021563DM
DRAW DATE: 05/15/07 08:08
PRINTED: 05/15/07 11:57
ACCESSION: 07135010

COMMENTS: FASTING
TESTS ORDERED: NOTE, CMP, URCA, DIG, TSH, LIPID

PROCEDURE	IN RANGE	OUT OF RANGE		REFERENCE RANGE	
NOTE:					
LAST DOSE TAKEN: PM, 5/14					
COMP. METABOLIC PANEL					
SODIUM	139			136-145	mEq/L
POTASSIUM	4.6			3.5-5.1	mEq/L
CHLORIDE	101			97-107	mEq/L
CARBON DIOXIDE	29			21-31	mEq/L
ANION GAP	14			10-20	
ALBUMIN	4.7			4.2-5.3	g/dL
PROTEIN, SERUM	6.5			6.0-8.3	g/dL
CALCIUM	9.7			8.6-10.3	mg/dL
BILIRUBIN, TOTAL	0.8			0.1-1.2	mg/dL
UREA NITROGEN, BLOOD		23	H	10-21	mg/dL
CREATININE, SERUM	1.2			0.6-1.3	mg/dL
ALK. PHOSPHATASE	62			41-111	U/L
ALT (SGPT)	42			0-46	U/L
AST (SGOT)	19			9-42	U/L
GLUCOSE		106	H	70-105	mg/dL
URIC ACID, SERUM		8.0	H	3.5-7.2	mg/dL
DIGOXIN	1.6			0.5-2.0	ng/mL
TSH	3.670			0.35-5.50	uIU/mL
LIPID PANEL					
CHOLESTEROL		262	H	90-200	mg/dL
HDL	36			>60 mg/dL Low Risk	
				40-60 Borderline/Moderate	
				<40 High Risk	
TRIGLYCERIDES		620	H	23-231	mg/dL
TC:HDL RATIO		7.3	H		

CHOLESTEROL (mg/dL)

	LDL	HDL	TOTAL
Desirable level/low risk	<130	>60	<200

END OF PAGE 1. CONTINUED ON PAGE 2

5/17 Appt please
apt 6/4 ww

NMS LABS

NMS Labs
3701 Welsh Road, PO Box 433A, Willow Grove, PA 19090-0437
Phone: (215) 657-4900 Fax: (215) 657-2972
e-mail: nms@nmslabs.com
Robert A. Middleberg, PhD, DABFT, DABCC, Laboratory Director

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June 24, 2008

TO: 60C
Santa Cruz County Coroner
Attn: Alan Burt
701 Ocean Street, #340
Santa Cruz, CA 95060

SUPPLEMENTAL TOXICOLOGY REPORT OF: **McCORNACK, Daniel E.** **45/M**

NMS Workorder No: 08095896
NMS Control No: 10843208
Client ID No: 08-0279[illegible]

SPECIMENS: One gray top tube containing ~ 10 mL of peripheral blood, one clear vial containing ~ 14 mL of peripheral blood and two white plastic containers (one containing ~ 30 mL of urine and one containing ~ 32 g of liver) were received on 03/28/08.

EXAMINATION: Analysis Requested - Panel 8092B - Autopsy Toxicology Therapeutic and Abused Drug Screen
Test No. 1615B – Digoxin

FINDINGS:

Blood

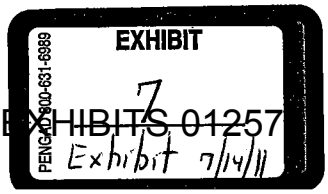
ETHYL ALCOHOL (by Enzymatic Assay & Headspace GC)	48 mg/dL (BAC=0.048 % w/v)
DILTIAZEM (by GC & GC/MS)	630 nanog/mL
DIGOXIN (by LC-MS/MS)	3.6 nanog/mL
QUINIDINE/QUININE* (by GC/MS)	Trace
ATROPINE (by GC/MS)	Positive

*Quinine and quinidine can be differentiated analytically, but this is a separate analysis. If further delineation is necessary, please contact the laboratory.

Incidental findings by GC/MS: CAFFEINE and THEOBROMINE.

Other than the above findings, examination of the specimens submitted did not reveal any positive findings of toxicological significance by procedures outlined in the accompanying Analysis Summary.

6-27-08



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NMS Workorder No: 08095896
NMS Control No: 10843208
Client ID No: 08-02790

COMMENTS:

1. Ethyl alcohol is a CNS-depressant and has effects so-related, e.g., impaired judgment, alertness and coordination.

   If the determined blood alcohol concentration (BAC) is representative of the circulating BAC at the time of the fatal incident, then it represents as absorbed body burden of approximately 2 "drinks" of an alcoholic beverage in an adult of average size weighing approximately 155 lbs.

   Note: a "drink" = 1 oz. of distilled spirits
   4 oz. of wine
   12 oz. of beer

   Each of the drinks listed above contains about the same amount of ethyl alcohol.

2. Diltiazem (Cardizem®) is a calcium channel blocking coronary vasodilator indicated for the treatment of variant, exertional and unstable angina. It is also used in arrhythmic and/or hypertensive therapy. Desacetyldiltiazem is an active metabolite of diltiazem. Divided doses up to 180-360 mg daily may be prescribed for angina.

   Therapeutic blood levels of diltiazem appear to be in the range of 50 to 200 nanog/mL. Numerous cases of diltiazem overdose have been reported. The majority of individuals who receive prompt treatment survive diltiazem overdose; however, death has been reported, especially in conjunction with other substances. Diltiazem has been found mixed with cocaine, either as a cutting agent or in an attempt to reduce cocaine-induced increased blood pressure. In a separate, small series of diltiazem related fatalities, the postmortem blood concentrations range from 6700 to 33,000 nanog/mL (mean 16,000 nanog/mL). In addition, diltiazem is reported to undergo postmortem redistribution with an average heart blood/femoral blood ratio of 2.6.

3. Digoxin (Lanoxin®) is a cardiac glycoside used in the treatment of congestive heart failure and other contractility-related deficiencies. There is considerable individualization of the dose of this medication and what is therapeutic in one individual may be toxic in another.

   Individuals are generally titrated to find an appropriate dosage, especially since digoxin has a low therapeutic index.

4. Quinine and quinidine are stereoisomers derived from the bark of the cinchona tree. Quinine has been used in the past as an antimalarial, but is more commonly used today to treat muscle cramps. It is also used as a flavoring agent in tonic waters and as a cutting agent adulterant in illicit street drug dosages of heroin. Adverse effects include gastrointestinal disturbances, tinnitus, dizziness, arrhythmias and hypotension.

   Quinidine is frequently used as an antiarrhythmic agent. It is available for acute administration by intramuscular or intravenous injection of 200 to 750 mcg or for maintenance therapy in oral doses of 600 to 4,000 mg daily. . Toxicity is manifested by gastrointestinal disturbances, giddiness, tinnitus, diplopia and hypotension.

5. Atropine is an anticholinergic alkaloid used in pre-anesthetic therapy to control airway secretions and as an antispasmodic to control gastrointestinal spasms. It is frequently used as an antidote in the treatment of anticholinesterase-type pesticides. It can be obtained naturally from deadly nightshade or jimson weed. Atropine is also used in resuscitative attempts.

   Toxic effects of atropine have considerable individual variation; however, at high doses, signs and symptoms include mydriasis, hot dry reddened skin, deliriums and hallucinations.

   In resuscitative failure, most of the administered drug remains confined to the intravascular injection pathway.

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NMS Workorder No: 08095896
NMS Control No: 10843208
Client ID No: 08-02790

Respectfully,

Edward J. Barbieri, Ph.D.
Forensic Toxicologist

EJB/ltb

This analysis was performed under chain of custody. The chain of custody documentation is on file at NMS Labs.

Unless alternate arrangements are made by you, the remainder of the submitted specimens will be discarded six (6) weeks from the date of this report; and generated data will be discarded five (5) years from the date of this report.

****** **** ANALYSIS SUMMARY **** ******

8092B - Therapeutic and Abused Drug Screen

Test No. 8092B – Drug Screen by Enzyme-Linked Immunosorbent Assay (ELISA) on Blood for: Amphetamine, Barbiturates, Benzodiazepines, Cannabinoids (Marihuana), Cocaine/Metabolites, Methamphetamine, Opiates and Phencyclidine (PCP); Headspace Gas Chromatography for Ethanol, Methanol, Acetone and Isopropyl Alcohol.

Test No. 8092B - Drug Screen II- Gas Chromatography and Gas Chromatography/Mass Spectrometry Analysis on Blood:

The following is a general list of compound classes included in the Gas Chromatographic screen. Other specific compounds outside these classes are also included. Please note that not all known compounds included in each specified class or heading are included. The detection of any particular compound is concentration-dependent. For a detailed list of all compounds included in this screen, please contact NMS Labs.

Analgesics (opioid and non-opioid), Anesthetics, Antiasthmatic Agents, Anticholinergic Agents, Anticonvulsant Agents, Antidepressants, Antiemetic Agents, Antihistamines, Antiparkinsonian Agents, Antipsychotic Agents, Antitussive Agents, Anxiolytics (Benzodiazepine and others), Cardiovascular Agents (non-digitalis), Hallucinogens, Hypnosedatives (Barbiturate and others), Muscle Relaxants, Non-Steroidal Anti-Inflammatory Agents (excluding Salicylate) and Stimulants (Amphetamine-like and others).

Test No. 8092B - Colorimetric Analysis on Blood for: Salicylates and Acetaminophen.

Test No. 5010B - Alcohol Confirmation - Enzymatic Assay on Blood for: Ethanol (Ethyl alcohol).

Test No. 1640B - Diltiazem - Gas Chromatography on Blood for: Diltiazem.

Test No. 1615B – Digoxin – Liquid Chromatography - Tandem Mass Spectrometry on Blood for: Digoxin.

******* END OF REPORT *******

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On April 25, 2008, Actavis Totowa® LLC, the manufacturer of Digitek® 0.125 mg and Digitek 0.25 mg tablets, issued a Patient Level Recall of all lots of these products as a precaution **because the tablets may be double the appropriate thickness and could contain twice the approved level of active ingredient.** Because of this, the manufacturer is recalling all lots of these products.

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If you filled a prescription for Digitek 0.125 mg or Digitek 0.25 mg tablets between **January 28, 2008** and **April 28, 2008,** we will be sending replacement product to you that is not affected by this recall at no cost to you. You will also receive instructions on how to return your remaining Digitek.

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Jan Berger, MD, MJ
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Estimating antemortem drug concentrations from postmortem blood samples: the influence of postmortem redistribution

D S Cook, R A Braithwaite, K A Hale

Abstract

***Aims*—To compare blood drug concentrations during life with postmortem drug concentrations measured from a peripheral site and a central site.**

***Methods*—Coroner's cases from October 1990 to July 1997 were reviewed. Six cases had data on both antemortem and postmortem blood drug concentrations. The postmortem to antemortem ratio was compared with the postmortem central to peripheral ratio, using cardiac blood as a central site and femoral blood as a peripheral site.**

***Results*—Drugs that have a high postmortem central to peripheral ratio; that is, drugs that exhibit considerable postmortem redistribution, also have high postmortem to antemortem ratios.**

***Conclusions*—A large degree of error can arise from attempting to estimate antemortem drug concentrations and the ingested dose from postmortem measurements. The chosen site and technique for postmortem blood sampling can greatly influence the concentration of drug measured.**

(*J Clin Pathol* 2000;53:282–285)

Keywords: postmortem blood sampling; drug concentrations; toxicological analysis

Postmortem drug redistribution refers to the processes by which the movement of drugs and other chemical poisons between tissues, organs, and body fluids takes place after death. This phenomenon is well recognised, and was first reported 25 years ago.[1] Since then, considerable effort has gone into elucidating the processes responsible.[2–16] Consideration of the redistribution of drugs is important in a variety of situations. Cases of suspected poisoning, either homicidal or suicidal; the role of drugs in "marginally toxic" cases, such as vehicle accidents; and also potential cases of euthanasia or medical negligence might rely upon the toxicological analysis of blood samples.[2] The timing, method of collection, and source of the sample might influence the interpretation of toxicological analyses.

The processes of postmortem redistribution result in the migration of drugs between blood and tissues. The rate and extent of this movement varies according to several factors, including the nature of the drug and the time interval between death and postmortem specimen collection. Within the torso, the major organs constitute potential drug pools, and the gastrointestinal tract might contain considerable quantities of unabsorbed drug, and thus central blood is subject to redistribution from these local organs. Peripheral blood, such as femoral blood, is subject to redistribution influences only from local tissues—muscle and fat. In general, redistribution into central vessels is greater than redistribution into peripheral vessels. The difference between the two sites is known as the central to peripheral (C/P) ratio. For these reasons, the blood specimen of choice for toxicological analysis after death is a femoral venous sample, ideally collected from a ligated vessel,[7 10] although inevitably there will be situations in which such sample collection is not possible.

Often, pathologists or toxicologists are requested to estimate the amount of drug present at the time of death, or the number of tablets consumed. This assumes that the drug concentration found at postmortem examination is a reliable estimate of that present at the time of death. There is a lack of evidence that such an extrapolation is possible; in only a few cases reported in the literature are antemortem blood concentrations available for comparison with values from a variety of sites at postmortem examination.[14 17–19] Such comparisons have not been carried out extensively because antemortem samples are often not available for analysis. In 1978, Vorpahl and Coe[17] collected data pertaining to antemortem and postmortem blood digoxin concentrations. The chosen postmortem blood sampling site was the left ventricle. Their results showed that in all 27 cases, postmortem cardiac blood concentrations were significantly higher than antemortem blood concentrations. Blood from the femoral vein was only collected in 11 cases; in these cases, the average postmortem to antemortem (PM/AM) ratio was 1.4, and in nine of the 11 cases the postmortem concentration was higher than the antemortem value.

Our paper comprises a series of brief case vignettes in which both antemortem and peripheral postmortem blood samples were analysed. The relation of the postmortem drug concentration to the antemortem concentration is investigated, and possible explanations for the variation seen are discussed in terms of postmortem redistribution.

Methods

We searched the coroner's archive of postmortem cases held at the regional laboratory for toxicology. The data were collected from October 1990 to July 1997, 950 cases in all. In six

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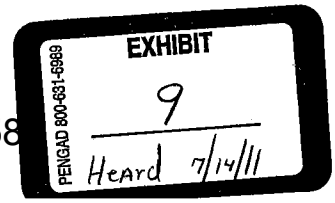

cases, both antemortem and postmortem blood samples labelled as "femoral blood" had been analysed, with data relating to seven drugs. All assays were performed on haemolysed whole blood with the use of internal quality assurance specimens for salicylate and paracetamol, and participation in NEQAS external quality assurance schemes for paracetamol, salicylate, amitriptyline, nortriptyline, and dothiepin. No internal or regular external quality assurance specimens were available for propranolol, methadone, and dextropropoxyphene during our study period. The methadone and dextropropoxyphene assays were validated by specimen exchange with other laboratories offering these analyses. In all cases except case 5, extensive drug screening was carried out. A review of the literature was also carried out to find reported C/P ratios for these drugs. Where a C/P ratio was not quoted in the literature, it was calculated for individual drugs from the central and peripheral postmortem blood concentrations determined for the case in question. In four of the cases (cases 2–5), the interval between antemortem sampling and death was known, and an estimate of the plasma concentration at the time of death was calculated using the following equation to account for the decline in drug concentrations as a result of ongoing metabolism and elimination during life[17]:

$$\ln N_t = \ln N_0 - \ln 2(T/t_{½})$$

where: N_t = calculated plasma drug concentration at time of death; N_0 = plasma drug concentration at time of sampling; T = time interval between sampling and death; $t_{½}$ = elimination half life of drug in plasma.[20]

Case histories

CASE 1

An 18 year old woman took an overdose of propranolol. She died shortly after admission to hospital; the postmortem examination was carried out on the next day. Propranolol was measured by means of reverse phase high performance liquid chromatography (HPLC) with ultraviolet (UV) detection.

CASE 2

A 22 year old man consumed a quantity of methadone mixture bought on the street. He was admitted unconscious to hospital and remained on life support for two days. Comprehensive drug screening for acidic, basic, and neutral drugs performed on an admission urine specimen detected only methadone and its major metabolite. Methadone was measured by means of reverse phase HPLC with UV detection.

CASE 3

A 74 year old man was admitted to hospital, having allegedly taken a paracetamol overdose. He died some 12 hours later. Paracetamol was assayed by means of an immunoassay (fluorescence polarisation immunoassay; Abbott Diagnostics, Abbott House, Maidenhead, Berkshire, UK). No other drugs were detected by screening capillary gas chromatography.

CASE 4

A 42 year old man was admitted after an overdose of dothiepin and distalgesic (paracetamol and dextropropoxyphene). He was maintained on a life support machine for one day. An antemortem blood sample was collected immediately before disconnection of this support. Both dothiepin and dextropropoxyphene were measured by means of reverse phase HPLC with UV detection, with simultaneous measurement of the major metabolites norpropoxyphene and nordothiepin.

CASE 5

A 28 year old woman was admitted having taken three overdoses of aspirin and Anadin in the previous three days. Antemortem blood was collected 4.3 hours before death. Salicylate was measured by colorimetry (Trinder's method). Quantitative immunoassay screening detected no tricyclic antidepressants.

CASE 6

A 41 year old woman was found unconscious having possibly taken an overdose of a friend's amitriptyline tablets. She was admitted to hospital but died the same day. Amitriptyline and nortriptylene were measured by means of reverse phase HPLC with UV detection. No other drugs were detected by screening capillary gas chromatography.

Results

Table 1 shows the data collected in the above cases. No further clinical, haematological, or biochemical data were available from the retrospective study of these cases.

The general trend of the data tabulated above is that drugs with a high C/P ratio also tend to have a high PM/AM ratio. In these examples, propranolol is a notable exception, having a PM/AM ratio lower than the C/P ratio. The elimination half life of propranolol is two to four hours.[20] In the interval between antemortem sampling and death, continued metabolism of the drug will lower the circulating concentration. After four hours, in most individuals, the circulating concentration will be half that measured previously, if not lower. The time between sampling and death was not known in this patient. If the interval between sampling and death is of this order, or greater, then considerable overestimation of the drug concentration at death will result. Overestimation of the antemortem concentration will result in an artificially low PM/AM ratio.

Table 1 Comparison of postmortem peripheral blood (PM)/perimortem (AM) plasma ratios with C/P ratios for a selection of drugs

Drug	*PM/AM*	*C/P‡*	*C/P range‡*
Dothiepin	11.7	2.2*	–
Dextropropoxyphene	8.3	3.2	0.9–7.3
Amitriptyline	3.9	2.9	0.9–13.9
Methadone	2.6	2.6*	–
Propranolol	1.9	2.5	1.0–3.8
Paracetamol	1.5	1.3	0.7–2.8
Salicylate	1.0	1.3	0.5–3.9

‡From Dalpe-Scott *et al*, 1995.[21]
*Calculated from the specific case.
C/P, central to peripheral ratio.

Discussion

These six cases illustrate that it can be dangerous to attempt to relate a drug concentration found at postmortem examination to the antemortem circulating concentration or to the antemortem dose received. In every case, the postmortem drug concentration in blood was as high as, or higher than, the antemortem circulating plasma drug concentration at the time of death. In several cases, the difference between the two concentrations was immense and cannot be accounted for by analytical error or differences in distribution of the drug between plasma and red blood cells. It is not possible to form a general rule regarding the difference between the two measurements, because the variation seen depends on the drug in question, as other authors have suggested.

The PM/AM ratios listed in the results table are all based on single case reports. It is possible that, if more cases were available, the PM/AM ratios found would be very different. The many factors that influence both C/P and PM/AM ratios, together with the wide variation of C/P ratios documented, suggest that such variations might also be encountered in PM/AM ratios. Thus, the PM/AM ratios listed cannot be applied in every case of death by these drugs.

In addition, other variables might influence the C/P ratio, such as the interval between death and postmortem examination. This is a factor that has been shown to affect site specific postmortem drug concentrations to a great extent,[16] and hence the C/P ratio calculated. It is likely that this interval will also affect the PM/AM ratios calculated for these drugs, although drug concentrations in the femoral vein after death appear to be relatively stable with time,[22 23] again making any extrapolation using these ratios unsafe.

Nonetheless, the trend of the data as a whole is striking. The finding that drugs exhibiting a high C/P ratio tend to have a high PM/AM ratio might prove useful in dealing with future cases in which poisoning may be a feature. The factors that influence postmortem redistribution are well established, and useful lists of C/P ratios have been published by a number of authors.[21 24]

It is often necessary to determine whether the drug concentration found at postmortem examination should be attributed to either therapeutic ingestion or overdose. This is very difficult to determine because of the influences of postmortem change. The use of PM/AM ratios, or back extrapolation from a postmortem concentration, is not recommended. For certain drugs, it may be more appropriate to consider the parent to metabolite ratio. It has been shown consistently for several drugs (for example, tricyclic antidepressants) that a high ratio is indicative of acute administration, as seen in overdose, because often in vivo metabolism is saturated or incomplete and circulating concentrations of the parent compound remain high.[25-27] It is necessary to consider whether the parent compound and its more polar metabolites are subject to differing degrees of postmortem redistribution themselves, further confusing the issue.

The difficulties arising from postmortem redistribution must be put into the context of working practice. The development of a standardised protocol for toxicological sample collection at postmortem examination can assist both the pathologist and the toxicologist in their work. An important feature of such a protocol is its practicability. The chosen method must be easily incorporated into routine postmortem practice. Blood for quantitative analysis (≈ 5 ml) should be taken from two distinct peripheral sites, preferably left and right femoral veins. Femoral blood can be taken by cutting the external iliac vein proximal to the inguinal ligament, and milking the distal cut end into the specimen tube. Early ligation of this vessel is recommended to avoid mixing with more central blood during evisceration.[7 10 28] An additional larger specimen of blood (≈ 20 ml) for qualitative screening can be collected from a named convenient large vessel. For deaths that have occurred in hospital, the hospital pathology laboratory should be contacted as soon as possible to see if any antemortem specimens of urine, blood, or plasma are available, and these should also be sent for analysis.

Our study shows that a high degree of error can arise from attempting to predict antemortem concentrations from postmortem concentrations, and emphasises the need for continued research into this area of pathology practice. In the absence of such data, estimates of circulating drug concentrations during life should not be made. In borderline cases where drugs might be involved, the toxicological findings should only be used to support known clinical or pathological findings.

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9-3

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9-4

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Review

Mechanisms Underlying Postmortem Redistribution of Drugs: A Review

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Abstract

Postmortem drug concentrations do not necessarily reflect concentrations at the time of death, as drug levels may vary according to the sampling site and the interval between death and specimen collection. These site- and time-dependent variations are called "postmortem redistribution" (PMR). The underlying mechanisms are complex and of different types. Passive drug release from drug reservoirs such as the gastrointestinal tract, liver, lungs, and myocardium may occur immediately after death and, later on, cell autolysis and the putrefactive process participate in redistribution. There is evidence that basic lipophilic drugs with a large distribution volume are particularly susceptible to PMR. Nevertheless, this cannot explain the actual PMR of some nonbasic, nonlipophilic drugs. In addition, the persistence of drug metabolism immediately after death must be considered. Consequently, it is of great importance to analyze specimens from different sampling sites in order to detect potential PMR and avoid misinterpretation of results.

Introduction

In forensic toxicology, the severity or lethality of a given intoxication is generally appreciated in the light of the blood concentration of the toxic compound (or "xenobiotic") involved, for which reference values such as therapeutic, toxic, or lethal levels often exist. Although, in the living, blood concentrations may allow evaluation of the total amount administered (e.g., following a single administration), taking into account the pharmacokinetic characteristics of the given molecule, this evaluation is generally not possible in the postmortem period. The main reason for this is that the concentrations obtained from postmortem samples do not necessarily reflect the blood concentrations at the time of death due to variations in these concentrations according to the sampling site and the interval between death and sampling. These variations, gathered under the generic term of "postmortem redistribution" (PMR), sometimes render the interpretation of results difficult, in as much as they concern molecules frequently involved in forensic toxicology such as opiates (1), amphetamines (2), cocaine (3), or tricyclic antidepressants (4).

This literature review shows that (1) certain organs are major sources of such PMR; (2) cell and tissue modifications during putrefaction are involved; and (3) the pharmacokinetic characteristics of the molecules are probably favoring factors, though knowledge is still limited as to the exact mechanisms involved.

Sources of Postmortem Redistribution

Many drugs are sequestered antemortem in organs qualified as "drug reservoirs" (Table I). After death, they are redistributed to the surrounding tissues. Hollow organs, such as different parts of the gastrointestinal tract, or viscera with a high concentrating power, such as the liver and lungs (the myocardium also), can be classed as drug reservoirs. PMR from these organs can occur by two different mechanisms: diffusion through blood vessels and transparietal diffusion towards the surrounding organs.

Redistribution from the gastrointestinal tract

Unabsorbed drugs in the stomach at the time of death can be redistributed to mediastinal vessels and surrounding organs according to the two mentioned mechanisms (Figure 1). Through the vasculature, the molecules rapidly diffuse to the left cardiac chambers, aorta, right cardiac chambers, and inferior vena cava (5,6). This diffusion can begin within hours after death, as described for ethanol (5) and tricyclic antidepressants according to Hilberg et al. (4) and Pohland and Bernard (7), who reported increasing heart blood concentrations of amitriptyline and fluoxetine with in the first 2 h after death.

Passive diffusion from the gastric content into surrounding organs mainly concerns the lower lobe of the left lung, the left posterior margin of the liver, and to a lesser extent the caudate lobe and—when the corpse is in a supine position—the posterior part of the right lobe (8). The pericardial fluid and the

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9-5

myocardium are also affected by redistribution from the stomach, and it is likely that the close anatomical apposition of the fundus against the diaphragm is a determinant factor (5). Diffusion to these sites was unambiguously reported for ethanol (5), amitriptyline, methanol, and lithium (8).

Contamination of airways by regurgitation of drugs from the stomach can at last induce the redistribution of these drugs into the pulmonary vessels and then into cardiac blood (9). Such contamination could result from inhalation during agony or passive relaxation of the esophagogastric sphincter contemporary to the advent of rigor mortis (9,10). This process may be facilitated by body handling and the supine position of the cadaver (5,10). This contamination of airways is associated with an increase in the drug blood concentration. Pounder and Yonemitsu (11) reported drug concentrations higher in blood from the aorta and superior vena cava than from the left and the right cardiac chambers, respectively, suggesting direct diffusion into these vessels rather than diffusion via the pulmonary and cardiac blood. This phenomenon was described for ethanol, paracetamol, and propoxyphene (11).

PMR concerns the whole upper gastrointestinal tract and not only the stomach (5). Moreover, it is influenced by physical factors such as drug concentration in the gastrointestinal content, the volume of this content, the temperature of the corpse, and time between death and sampling. As expected, it is slowed by refrigeration at 4°C and increases with the delay between death and autopsy (5). Finally, it is worth noting that pre-existing pathological conditions may influence PMR, as suggested by Pounder and Smith (5), who described a much lower redistribution from the gastric content towards the pericardium and mediastinal blood vessels in a patient suffering from a large adenocarcinoma of the left lung basis than in an individual deceased because of myocardial infarction.

Table I. Mechanisms for Postmortem Redistribution

Mechanisms	Consequences
Drug reservoirs	**Redistribution to surrounding tissues**
Gastrointestinal tract	Cardiac chambers, thoracic vessels, left lung, liver, inferior vena cava
Lungs	Cardiac chambers, thoracic vessels, liver
Myocardium	Heart blood
Liver	Inferior vena cava, right cardiac chambers, pulmonary vessels, stomach, duodenum, gall bladder
Cadaveric changes	
Cell death	Leakage of xenobiotics into the extracellular space
Blood coagulation and hypostasis	Modification of serum/blood ratio
Blood movements	Transport of xenobiotics and mixing of bloods from different origins
Putrefactive process (bacteria)	Degradation and/or synthesis of macromolecules
Drug chemical and pharmacokinetic properties	
Acidic/basic properties	?
Lipophilicity	?
Drug binding proteins or red cells	?
High volume of distribution (Vd)	Leakage from tissues
Residual metabolic activity	?

Redistribution from the lungs

In vivo, the lungs receive the entire blood flux from the right ventricle and so accumulate many drugs, particularly weak lipophilic bases with pK_a values greater than 8, such as imipramine, amitryptiline, methadone, or chlorpromazine (12–14). PMR from the lungs begins within the first two hours after death, inducing a rise in drug concentrations in cardiac chambers and thoracic vessels (4,6,11,13,15), and seems to be more intense than redistribution from the gastrointestinal tract. In all the cases reported, the concentrations in the aorta and left cardiac chambers were higher than in the superior vena cava and right cardiac chambers (16). The two previously described mechanisms are probably involved in this redistribution (Figure 2).

First, the drugs can be redistributed via the pulmonary vessels. The earlier postmortem increase in drug levels in pulmonary veins than in the arteries could result from the fact that diffusion through the thinner-walled veins occurs more rapidly (11,17,18). On the other hand, drugs sequestered in lung parenchyma and vessels could be redistributed directly into surrounding tissues, including thoracic vessels and cardiac chambers (19).

The fact that concentrations in the aorta and the superior vena cava were higher than in the left and right cardiac chambers, respectively, suggests direct redistribution into both the aorta and inferior vena cava from surrounding tissues, that is, from the left lung, left primary bronchus, and carina for the aorta and from veins adjacent to the trachea for the superior vena cava (11).

The intensity of the redistribution from lungs could be explained by the large surface area of the alveoli, the thin diffusion membrane, and the high vascularization (20). This redistribution may coexist with redistribution from the gastrointestinal tract, and it could be very difficult to determine the main mechanism when drugs were taken orally. According to Hilberg et al. (19), when drug concentrations are higher in the heart blood than in the myocardium, diffusion from the lungs via the pulmonary veins must have occurred. On the other hand, when drug concentration in the myocardium is higher, diffusion directly from the stomach or lungs through the myocardium is a reasonable explanation. According to Pounder (10), when heart blood concentrations are higher than concentrations in the pericardial fluid, redistribution must come from the lungs, and the opposite result indicates that redistribution comes from the stomach.

Finally, PMR from the lungs to the liver has been described. The anatomical frontier between the thoracic and abdominal cavities is the diaphragm. However, if diffusion across this barrier was the only source of this redistribution, one could expect higher drug concentrations in the liver segments in contact with the diaphragm, which is not the case. The pleural and peritoneal fluids are regarded as vehicles for drug exchanges between the organs and the walls of the two anatomical cavities (15). This hypothesis was confirmed in humans (6) and animals (15).

9-6

Redistribution from the liver

PMR from the liver is complex, as it involves different mechanisms. Drugs sequestered in the liver at the time of death could be redistributed via the hepatic veins to the inferior vena cava and then into the right cardiac chambers and pulmonary vessels or to peripheral venous blood (20). One of the results of this redistribution is the decrease in drug concentrations in hepatic lobes, as described by Pohland and Bernhard (7) for fluoxetine and norfluoxetine. Nevertheless, this process is not as intense and early as redistribution from the lungs.

Secondly, drugs could be redistributed directly into adjacent organs. The anatomical relationships of the human liver that are relevant to PMR are with the stomach, the pylorus, the proximal duodenum, and the gall bladder (20), but this is not as important as the redistribution via the hepatic vessels.

The liver is also the target of redistribution from the gastrointestinal tract. The anatomical relationships between the digestive tract and liver are of great importance for the understanding of these mechanisms. The greatest part of the inferior surface of the left liver lobe is in contact with the stomach, and the pylorus and proximal duodenum rest against the right lobe of the liver and the gall bladder. Finally, taking into account the close anatomical proximity between the liver and the stomach, the xenobiotics contained in the latter at the time of death can enter the hepatic parenchyma either by passive diffusion or through the portal vein (20). This diffusion phenomenon is very variable, with the left hepatic lobe, which is in close contact with the stomach, being more involved than the others. Accordingly, Pounder and Davis (21) found much higher concentrations of zopiclone in the left lobe and in the gall bladder than in the right lobe. Fuke et al. (6) demonstrated, in a human cadaver model, that after gastric instillation of 25 mL toluene and isobutanol, the concentrations measured in the left lobe were markedly higher than those in the right. It is therefore particularly difficult to correctly interpret the evolution of hepatic drug concentrations postmortem. Following Hilberg et al. (22), the molecular pK_a could have an influence on these different mechanisms. It would be interesting to find such general rules, as redistribution from or towards the liver concerns a very large number of molecules, an exhaustive list of which is impossible to establish.

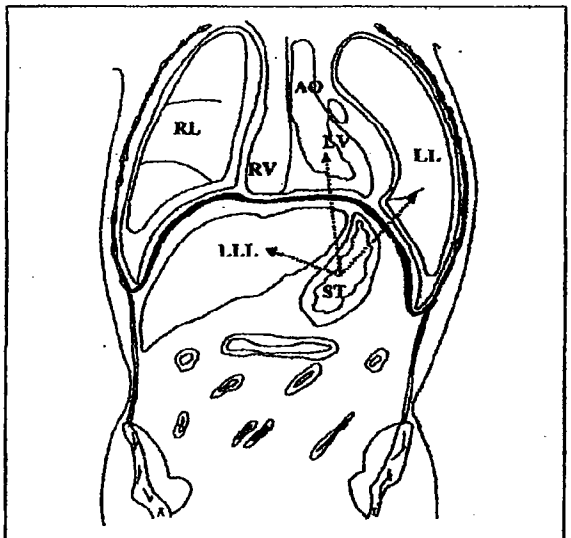

Figure 1. Frontal view of thorax and abdomen through the left ventricle. The arrows show the direction of redistribution towards the main organs concerned (RL, right lung; LL, left lung; AO, aorta; RV, right ventricle; LV, left ventricle; ST, stomach; and LLL, left liver lobe).

Redistribution from the myocardium

In the living, many cardiac drugs are concentrated in the myocardium. One of the best examples is digoxin, with in vivo myocardic concentrations 30 times higher than that in heart blood. This is also the case for calcium channel blockers and quinidine. Rapidly, these drugs are redistributed into cardiac blood, in which concentrations rise dramatically. A moderate increase has also often been described in the subclavian venous blood, which cannot, for this reason, be considered as a peripheral blood specimen (23). Such redistribution from the myocardium into heart blood has also been described for other drugs such as morphine (24,25); amphetamine (23,26); methamphetamine (13,26); propoxyphene and norpropoxyphene (23); imipramine and desipramine (27); and amitriptyline, doxepin, maprotiline, and metoprolol (23). Because the dramatic concentration increase observed in cardiac blood may result from redistribution from the stomach, lungs, or liver, proof of redistribution from the myocardium as the primary mechanism responsible for this increase would be demonstrated by higher concentrations of the drug in both the left and right cardiac chambers (18).

Redistribution into body fat

Conversely to the previously described mechanisms, some very highly lipophilic drugs are concentrated in adipose tissues by simple physical dissolution in neutral fats. This distribution

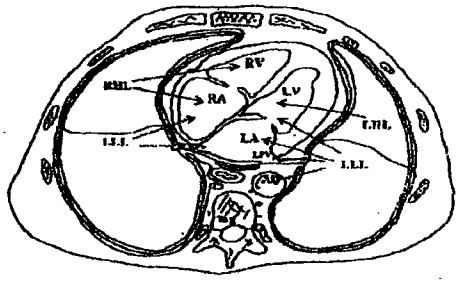

Figure 2. Cross-section of thorax through the 8th thoracic vertebra (caudal view). The arrows show the direction of redistribution from the lungs into the organs concerned. Diffusion occurs via the pulmonary veins (LPV, left pulmonary vein and RPV, right pulmonary vein) and transparietally towards surrounding anatomic structures (LA, left atrium; LV, left ventricle; AO, aorta; LHL, left higher lobe of lungs; LLL, left lower lobe of lungs; RHL, right higher lobe of lungs; RLL, right lower lobe of lungs; RA, right atrium; and RV, right ventricle).

9-7

occurs slowly, as the blood flow to adipose tissues is low and the equilibrium between blood and adipose tissue concentrations may not have been achieved at the time of death (20). In such a case, a continued distribution (rather than a real redistribution) of these drugs from blood into adipose tissues, lowering the postmortem blood concentrations, can occur. This phenomenon occurs for anesthetics and volatile compounds (6,20).

Agonic and Cadaveric Changes

Cell death

A prevalent hypothesis to explain the release of basic drugs from solid tissues into the blood compartment within hours after death is that the decrease in blood pH would be responsible for a return of the basic molecules into this compartment, due to an increased concentration gradient in nonionized forms (Table I). Actually, it involves much more complex mechanisms, in as much as it is now clear that intracellular pH decreases before plasma pH, which is responsible for an inverse gradient in nonionized forms for basic molecules. The molecular mechanisms responsible for cell death are complex. Hypoxia due to the loss of oxygen-carrying capacity of blood during the agonic processes is the first stage. It produces a loss of oxidative phosphorylation capacity and halts the synthesis of adenosine triphosphate (ATP) by the aerobic pathway, inducing an increased rate of anaerobic glycolysis to provide the cell with some energy (28). Glycogen is, thus, rapidly depleted, and this anaerobic process results in the accumulation of lactic acid and inorganic phosphates that reduce the intracellular pH and, probably at this step, induces an increased accumulation of basic drugs into the cells.

The second consequence of this loss of ATP production—and consequently ATPase activity—is the failure of the energy-dependent sodium pump, which causes sodium to accumulate intracellularly with diffusion of potassium out of the cell. The net gain of solute is accompanied by an iso-osmotic gain of water resulting in cell swelling (28). The next phenomena to occur are detachment of ribosomes from the granular endoplasmic reticulum, dissociation of polysomes into monosomes, vacuolization of the mitochondria, and massive calcium cell influx. Finally, intracellular acidification and changes in ionic composition lead to damage to the lysosomal membranes with leakage of their enzymes into the cytoplasm and activation of their acid hydrolysis (28). Activation of these enzymes leads to enzymatic digestion of cell components and membranes. There is then a widespread leakage of cellular enzymes and macromolecules into the extracellular space. The basic lipophilic drugs, highly concentrated in the cells, are redistributed at this stage into the extracellular space and then into the blood compartment. Neutral or acidic drugs are less affected. According to Langford and Pounder (29), the release of enzymes into the extracellular space could be used as an indicator for PMR. They also demonstrated that there is a strong positive correlation between individual amino acids (glycine, leucine, methionine, serine, and valine) and drug concentrations in pulmonary blood samples. These autolytic processes concern all cells and tissues, but at different rates. The disintegration of physiological and anatomical barriers such as vascular walls leads to changes in drug concentrations. Skopp et al. (30) elaborated an in vitro experiment for studying postmortem vascular permeation. They demonstrated that in veins, sampled according to the selection criteria (corpses stored at 4°C within 6 h after exitus and autopsy performed within 24–60 h after death), concentrations of morphine and its glucuronides in the extravascular space increased with time during the first 120 h postmortem, according to a sigmoidal curve. In addition, the molecules were absorbed in part by the vascular wall.

Furthermore, vascular permeation could be strongly influenced by the initial substrate concentration, the molecular structure (i.e., the size, shape, charge, and partitioning behavior), and the orientation of solute flux. Finally, the corpse temperature appeared to have a large influence on this process: preservation at 4°C for the first two days tended to limit it, but it increased beyond 48 h postmortem, whatever the temperature of the corpse (30).

Blood coagulation and hypostasis

Postmortem blood sediments and clots unevenly in the body. The transformation is brought about by blood clotting followed by lysis (Table I). The two processes may occur simultaneously, and the effectiveness of the clot lysis will determine whether the blood is clotted or completely fluid, or partly clotted and partly fluid. This phenomenon varies from case to case (20). The clot generally entraps a large number of red blood cells, so sampling this clot for toxicological analysis may influence the concentration measured for any drug exhibiting unequal distribution between red blood cells and serum.

A few hours after death, hypostasis occurs by sedimentation of blood and plasma to the lower parts of the corpse. Hypostasis induces variations in the percentage of erythrocytes by volume, depending on the part of the body. Thomsen et al. (31) demonstrated that this percentage decreased by more than 50% during the first 9 h postmortem in blood samples taken from the cubital vein on the upper side of the arm. The position of the corpse was unfortunately not specified in this study.

Blood movements

Movements of blood within the vessels occur early after death and may be responsible for physical redistribution of drugs between different vascular compartiments (Table I). The extent of these postmortem blood movements is influenced by pressure and fluidity changes (20). Rigor mortis, which occurs during the first 24 h postmortem, induces systolic ventricular contractions with small movements of cardiac blood into the superior vena cava and the associated neck veins (32). With the increase in intra-abdominal pressure there is a blood reflux from the abdominal aorta into the thoracic aorta, from the inferior vena cava into the right atrium and superior vena cava, and from the left cardiac chambers into the pulmonary veins (20,32).

Later, at the onset of the putrefactive process and the disparition of rigor mortis, putrefactive gases distend the abdominal walls and the diaphragm, inducing a reflux of blood in peripheral veins which become more apparent. This phenomenon, incorrectly called "postmortem circulation", is of weak ampli-

tude. Furthermore, to our knowledge, the only experimental work designed to investigate this phenomenon dates back to 1961 (33) and should be brought up to date by studying the postmortem diffusion of autoradiographic blood flow tracers. Finally, the only experimental work dedicated to this phenomenon in animals demonstrated that, in rabbits, these movements were related to the cadaver position and highly variable from case to case (34).

Putrefactive processes

Putrefaction is highly variable depending on the ambient conditions and the state of the corpse. As part of this process, degradation and/or synthesis of xenobiotics by bacteria are possible, as previously described for ethanol (Table I).

Bacteria present in the gastrointestinal tract at the time of death are known to cross the gastrointestinal wall after death, enter the blood and lymph vessels, and then transmigrate throughout the body. This migration may occur within the first few hours postmortem, especially when ambient temperature is high. The bacteria most likely involved is this process are those originating from the gastrointestinal tract such as *Bacillus* spp., *Pseudomonas* ssp., *Escherichia coli*, *Proteus mirabilis*, *Clostridium perfringens*, *Staphylococcus epidermidis*, *Streptococcus fæcalis*, and *Bacteroides fragilis* (35–37). These bacteria could produce and/or metabolize many compounds in the postmortem blood. The best example is ethanol synthesis. In the presence of glucidic substrates, such as glucose or ribose, and amino-acids from protein breakdown, bacteria and yeasts can produce ethanol (36).

According to O'Neal and Poklis (35), *Escherichia coli* and *Candida albicans* are the primary causes of postmortem ethanol synthesis, but there are 58 species of bacteria and 17 species of yeasts that can produce ethanol. Kupfer et al. (38) developed a robust polymerase chain reaction (PCR) method for the rapid detection of *Escherichia coli*, *Proteus vulgaris*, and *Candida albicans* in human postmortem blood, in order to substantiate ethanol neo-formation when interpretation of results is difficult.

Because glucose is the primary substrate of postmortem ethanol production, the tissues with high glucose storage capacity are the sites of greatest ethanol synthesis: mainly the liver, skeletal muscles, lungs, and myocardium (35). Urine seems to be a poor medium for microbial ethanol synthesis, unless the deceased was diabetic (35,39). The brain would be concerned to a lesser extent: Davis et al. (40) reported that it was not until the third day postmortem that ethanol concentration exceeded the 0.01% detection limit usual in forensic analysis. As a result of microbial production, there is a wide variability in ethanol concentration between the sampling sites (41). According to Jones et al. (42), mannitol administration just before death could favor postmortem ethanol synthesis. Other volatile compounds such as methanol, *n*-propanol, isopropanol, *n*-butanol, and *sec*-butanol (43) may also be produced in postmortem blood during this process. The presence of these compounds can be used as a marker of postmortem ethanol synthesis (44,45). Taking into account these phenomena, most of the authors recommended sampling vitreous humor in order to distinguish between exogenous absorption and postmortem synthesis. Vitreous humor is considered an ideal specimen for the interpretation of postmortem ethanol concentration because (1) it contains no glucose or microorganisms, (2) it is protected from putrefaction or trauma, and (3) the concentration of ethanol in this medium reflects (is not equal because of differences in water content) the antemortem blood concentrations. Otherwise, ethanol can be metabolized by oxidation to acetaldehyde and acetate. Some bacteria and yeasts with fermentative and/or oxidative metabolisms, such as *Candida albicans* or *Serratia marcescens*, are able to produce and/or metabolize ethanol depending on the chemical properties of the medium (46), whereas bacteria with strict oxidative metabolisms, such as *Pseudomonas* spp, can only metabolize ethanol (37). The chronological aspects of ethanol synthesis and degradation is under debate. Takayashu et al. (47) studied the metabolism and postmortem changes of deuterium-labeled ethanol (ethanol-d_6) in rabbits. They demonstrated that the ethanol-d_6 concentrations decreased moderately in heart, liver, lungs, kidney, and femoral skeletal muscles during the first 90 min after death, and nondeuterated ethanol and 1-propanol concentrations showed marked increases depending on the degree of putrefaction of each organ or tissue, suggesting that ethanol precedes neo-formation. In contrast, according to Bouillerot and Laviano-Rousselin (37), ethanol degradation should occur after ethanol synthesis because of substrate depletion.

Postmortem degradation by bacteria does not only concern ethanol, some other compounds, such as benzodiazepines, could be metabolized by microorganisms during the putrefactive process. According to Robertson and Drummer (48), the nitrobenzodiazepines clonazepam, nitrazepam, and flunitrazepam are metabolized to their respective 7-amino metabolite in the liver, lungs, myocardium, kidneys, and skeletal muscles by enterobacteria (*Escherichia coli*, *Bacillus* spp, *Proteus mirabilis*, *Clostridium perfringens*, *Staphyloccocus aureus*, *Staphyloccocus epidermidis*, *Streptoccocus fæcalis*, and *Bacteroides fragilis*) containing an oxygen-sensitive nitroreductase enzyme able to reduce nitroaromatic compounds. As expected, this process is slowed down by keeping the corpses at + 4°C. The hypothesis of cyanide degradation by bacteria during the putrefactive process has been evoked. Ballante et al. (49) described a significant decrease in cyanide concentration with time in postmortem blood and tissues (lungs, brain, liver, and kidneys) of rabbits killed with potassium cyanide. This may be due to the metabolization of cyanide to thiocyanates, but the bacteria involved are unknown.

Pharmacokinetics

The PMR process may be influenced by the pharmacokinetic behavior of drugs. First, it should be stated that in vivo, many drugs present marked differences in blood concentrations with respect to the sampling sites. Arterial concentrations are often higher than venous concentrations during the absorption phase, whatever the mode of administration, as has been described for ethanol, diazepam, or phenobarbital (23,50). During

the elimination phase, the venous concentrations of drugs such as furosemide, procainamide, and propranolol are higher than the arterial concentrations. According to Chiou (50), drugs with a short half-life and/or a large apparent Vd may present more marked arteriovenous differences than drugs with a long half-life and/or a smaller Vd. The possibility that such arteriovenous differences at the time of death may influence PMR of the molecules concerned should be mentioned. This phenomenon was hypothesized by Elsirafy et al. (51) to explain differences in postmortem concentrations of diazinon between different sampling sites.

PMR of drugs depends not only on changes in cells and tissues during and after death, but also on the pharmacokinetic properties of these drugs (Table 1). Most studies have tried to link the intensity of PMR of a drug to its in vivo distribution, that is, its Vd. But the postmortem changes in pharmacokinetics are probably more complex, and modifications may occur at each pharmacokinetic stage, that is, absorption, distribution, metabolism, and elimination.

Absorption

There are many different processes by which a drug can cross the lipidic membranes of cells. Most drugs cross membranes by passive diffusion, ruled by Fick's law, depending on the concentration gradient of the molecule and the pH on both sides of the membrane; molecular size, lipid solubility, and the ionization state of the molecule; and on the pH on the both sides of the membrane.

Although no study has been conducted on the postmortem changes in drug absorption, it is more than likely that passive diffusion is affected by the postmortem changes of intracellular pH and by the loss of the membrane integrity. These changes may influence the absorption of drugs present in the gastrointestinal tract at the time of death, but pH changes along the digestive tract after death are unknown.

Filtration is another absorption process which concerns small, water-soluble molecules such as ethanol. These molecules cross the lipid membrane through proteic pores because of an osmotic or oncotic gradient. Equilibration of water and electrolytes on both sides of the membrane and damage to the proteic pores early after death tend to accelerate the filtration process at first and then stop it when equilibrium is reached.

In addition, some drugs, essentially weak organic acids and bases, cross the lipid membranes by active transport. Active transport requires energy input, often in the form of ATP, as well as the presence of a carrier system to transfer the molecules across the membrane. The cessation of ATP production rapidly after death probably blocks this process.

Distribution

After absorption, drugs reach the circulation in order to be distributed throughout the body and reach their site(s) of action.

Blood transport of xenobiotics. The first stage of distribution is the transport of xenobiotics in the bloodstream. In the circulating blood, drugs can be (1) dissolved in the plasma water, (2) bound to the plasma proteins, and/or (3) bound to the membranes or contained in the cytoplasm of blood cells (mainly erythrocytes). Only water-soluble molecules can be completely or almost completely dissolved in plasma water. This is the case for ethanol, for example, which is dissolved in total body water and whose PMR is not influenced by changes in blood proteins or red cells.

Drug protein binding occurs in different ways. It may take the form of either weak, hydrophobic bonds between lipid soluble xenobiotics and the hydrophobic sites of albumin and/or lipoproteins, or stronger electrostatic bonds between ionized, weak organic acids (e.g., sulphonamides) and the cationic sites of albumin or between weak organic bases (e.g., β-blockers, tricyclic antidepressants) and acid α_1-glycoprotein. Irreversible, covalent bonds between plasma proteins and certain molecules (e.g., antimalarial drugs) are also possible (52). The total protein blood concentration decreases after death because of the breakdown into amino acids and peptides resulting from acute anoxia (i.e., stopped synthesis) and from proteolysis by autoenzymes (53). This process would be accelerated at the beginning of putrefaction by proteolytic bacteria (54).

Oemichen et al. (55) reported the possibility of PMR of serum albumin from the intravascular space into the perivascular space. Even if no more recent studies have been devoted to the understanding of these postmortem changes, the rapid decrease in protein binding is evidence. The major result of this process is an increase in the intravascular concentration of the free form of the drugs, which could theoretically influence their redistribution. No information could be found in the literature about the potential postmortem changes in acid α_1-glycoprotein and lipoproteins. However, as postmortem drug levels are generally analyzed in whole blood, modifications in drug binding have little consequence for the interpretation of the analytical results.

Some drugs are bound to the red cells in the circulating blood. The postmortem whole blood concentration of these drugs could be affected by the process of hypostasis, inducing variations in the apparent hematocrit (1). This phenomenon was hypothesized by Tomson et al. (56) to explain PMR of phenytoin and by Skopp et al. (57) for morphine glucuronides. This is, however, not the sole explanation for all the postmortem concentration variations observed (58).

Distribution to solid tissues and organs. Antemortem distribution of a given drug is a dynamic process depending on its physicochemical properties (e.g., molecular size, degree of ionization, and lipophilicity) and physiological properties such as the blood flow perfusing the organs and the affinity of this drug for the different tissues or organs.

Some lipophilic drugs are known to have a very wide distribution in tissues as a result of cellular uptake and accumulation. There are two probable mechanisms: binding to membrane phospholipids and accumulation in acidic compartments (14). Under physiological conditions, plasma pH is approximately 7.4, and the pH of cells is of the order of 5. Weak bases, mainly in their nonionized state in the plasma, permeate cell membranes because of this pH gradient and accumulate extensively in lysosomes. This process of lysosomal trapping contributes to the uptake of the weak bases by different tissues such as the lungs, brain, heart, kidneys, and liver (12). The early pH changes

may explain the relase of these weak bases into the vascular compartment.

The distribution of a drug in the body is characterized by its apparent Vd, which refers to the volume into which the total amount of the drug would have to be uniformly distributed to reach the concentration actually measured in plasma. The value obtained for the Vd may or may not have physiological significance. Some studies suggest that drugs with a large Vd are those that are most widely redistributed after death because of their accumulation in tissues (59). This is obviously the case for weak lipophilic bases. Consistently, PMR was extensively reported for the tricyclic antidepressants such as amitriptyline (4), nortriptyline (18), trimipramine (60), or dothiepine (61,62). Similar results were found for fluoxetine (7), venlafaxine (63), methadone (64,65), amphetamine and methamphetamine (13), and even digoxine (66). According to Hilberg et al. (67), an apparent Vd of more than 3–4 L/kg is a good indication that the drug is liable to undergo PMR. But this hypothesis is not enough to explain the postmortem changes observed with many different types of molecules with different physicochemical properties. The case of the postmortem changes in blood levels of morphine and its glucuronides is of great interest. According to Sawyer and Forney (24), free and total morphine concentrations in rats increased significantly during the postmortem period, depending on the sampling site, with significant increases in cardiac blood, cardiac tissue, and liver, and on the postmortem delay, with highest concentrations at 96 h postmortem. The swiftness of the observed redistribution did not support the hypothesis of bacterial hydrolysis of the glucuronides into morphine. However, the authors present no precise data on the potential source of such a redistribution. Moreover, these results were not completely confirmed in humans: according to Logan and Smirnov (26), who studied the PMR of morphine and its glucuronides in 32 deaths involving opiates, no evidence was found for changes in morphine levles with time at either central peripheral blood sites. However, they confirmed that morphine concentrations were higher in cardiac blood compared to peripheral blood at every sampling time, particularly when the ventricular blood concentration exceeded 0.3 mg/L. The fact that the corpses were stored at 4°C during the experiments and that blood samples were drawn from the left ventricle may only partly explain the discrepancy between these results and those of Sawyer and Forney (24). Finally, it should be kept in mind that the anatomical relationships between abdominal organs are different in humans and rats and that the latter have no gallbladder. Gerostamoulos et al. (68) confirmed, in 40 cases of heroin-related death, that there was no statistically significant difference in concentrations with respect to the postmortem interval, but observed a trend for higher concentrations in central than in peripheral blood. Nevertheless, other authors have also described variations in morphine and morphine glucuronides concentrations depending on the sampling sites (69,70). However, the pharmacokinetic properties of morphine are quite different from those of the molecules that typically undergo PMR. Morphine is not a weak base as described, but an amphoteric compound which becomes less lipophilic with decreased pH, which may explain its redistribution from lipophilic tissues after death (24). However, several other hypotheses have been raised to explain this redistribution. According to Skopp et al. (1), the hypostasis process, resulting in plasma loss and hemoconcentration in the lower areas of the body, could play a part in the observed site-to-site variations (but this should also be the case for any drug or toxic compound dissolved in plasma, which is not likely). According to Carrupt et al. (71), morphine glucuronides can exist in two conformational forms, the folded one being more lipophilic than the unfolded one. The site-to-site variations in morphine glucuronide concentrations could be associated with this particularly. However, no single explanation currently prevails.

The PMR of acetaminophen is worth mentioning. Acetaminophen has a small apparent Vd of about 1 L/kg (72), low lipid solubility, and no known preferential tissue binding. Gomez et al. (73) administered 160 mg/kg of acetaminophen by oral gavage to rabbits and found site- and time-dependent changes in acetaminophen postmortem blood concentrations, with higher concentrations in cardiac blood compared to femoral blood at each sampling time, and an increase in cardiac blood concentrations over time. But the interpretation of these results must take into account the fact that the animals were sacrified only 20 min after the gavage, so it is likely that the antemortem distribution of acetaminophen was not complete at the time of death and that unabsorbed amounts of acetaminophen in the stomach could have diffused into the cardiac chambers. Such site-dependent variations were described in a case of lethal, acute acetaminophen intoxication (74), but were not found in a case of multiple-drug ingestion, indicating the possibility of a drug-drug interaction (27). The cause of acetaminophen redistribution remains unclear, and the possibility that the dose absorbed may influence this process cannot be ruled out. As acetaminophen undergoes extensive hepatic metabolism, the hypothesis of redistribution from the liver might be explored in animal models, for example by administering paracetamol by a parenteral route and collecting liver samples at different times, in order to look for a potential decrease in paracetamol concentration parallel to the increase in blood levels, as reported by Pohland and Bernard (7). The opposite situation is exemplified by mirtazapine, a relatively new tetracyclic antidepressant with basic and lipophilic properties and an apparent Vd of 4.84 L/kg (75), which did not exhibit any significant difference between cardiac and femoral postmortem blood concentrations, but showed an increase in liver concentrations (76,77). All these examples demonstrate that the postmortem site- and time-dependent variations of drug concentrations are not automatically associated with molecules with a high apparent Vd. Obviously, the absorption route must be taken into account, as diffusion from the gastrointestinal tract may concern all the drugs. Otherwise, drugs with a small apparent Vd can have a high affinity for some tissues from which they can be redistributed during the postmortem period (78). The liver and the lungs are obvious candidates, but probably also fatty tissues or skeletal muscles. Thus, paracetamol could be progressively redistributed from the liver parenchyma into the central blood vessels without affecting the peripheral vessels. It is worth noting that the brain, though highly vascularized, is less subject to PMR than the organs mentioned: studies in rats (24,67) have shown that the brain concentrations in